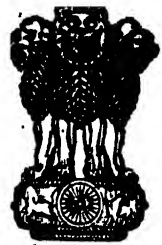

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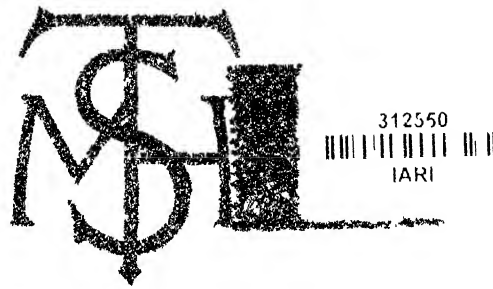
Vol. X. No 1. NOVEMBER, 1907

TRANSACTIONS

OF THE

SOCIETY OF TROPICAL MEDICINE AND HYGIENE.



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For Publishing Notice see page ii.]

TRANSACTIONS OF THE SOCIETY OF TROPICAL MEDICINE AND HYGIENE.

NOVEMBER, 1916. VOLUME X. NO. 1.

CONTENTS.

SPECIAL NOTICES.

1. The next Meeting of the Society will be held on Friday, the 17th November, at 5.30 p.m. As the afternoon Meetings seem to suit most Fellows, they will be continued until the War is over.

Tea will be provided for Fellows between 5 p.m. and 5.30 p.m.

2. Owing to the fact that the addresses of so many Fellows are still uncertain, and will be so until the War is over, no Year Book will be issued again this year. Fellows will therefore have to rely upon the one issued for 1914-1915 for the present.

EDITORIAL NOTICES.

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TRANSACTIONS OF THE SOCIETY OF TROPICAL MEDICINE AND HYGIENE.

DECEMBER, 1916. VOLUME X. NO. 2.

CONTENTS.

SPECIAL NOTICES.

1. The next Meeting of the Society will be held on Friday, the 19th January, at 5.30 p.m. As the afternoon Meetings seem to suit most Fellows, they will be continued until the War is over.

Tea will be provided for Fellows between 5 p.m. and 5.30 p.m.

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TRANSACTIONS OF THE SOCIETY OF TROPICAL MEDICINE AND HYGIENE.

JANUARY, 1917. VOLUME X. NO. 3.

CONTENTS.

SPECIAL NOTICES.

1. The next Meeting of the Society will be held on Friday, the 16th February, at 5.30 p.m.

Tea will be provided for Fellows between 5 p.m. and 5.30 p.m.

2. Owing to the fact that the addresses of so many Fellows are still uncertain, and will be so until the War is over, no Year Book will be issued again this year. Fellows will therefore have to rely upon the one issued for 1914-1915 for the present.

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TRANSACTIONS OF THE
SOCIETY OF TROPICAL MEDICINE AND HYGIENE.

FEBRUARY, 1917. VOLUME X. NO. 4.

CONTENTS.

SPECIAL NOTICES.

1. The next Meeting of the Society will be held on Friday, the 16th March, at 5.30 p.m.

Tea will be provided for Fellows between 5 p.m. and 5.30 p.m.

2. Owing to the fact that the addresses of so many Fellows are still uncertain, and will be so until the War is over, no Year Book will be issued again this year. Fellows will therefore have to rely upon the one issued for 1914-1915 for the present.

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TRANSACTIONS OF THE SOCIETY OF TROPICAL MEDICINE AND HYGIENE.

MARCH, 1917. VOLUME X. NO. 5.

CONTENTS.

SPECIAL NOTICES.

1. The next Meeting of the Society will be held on Friday, the 18th May, at 5.30 p.m.

Tea will be provided for Fellows between 5 p.m. and 5.30 p.m.

2. The Secretaries hope to be able to issue a new Year Book this summer. They specially call the attention of Fellows to this fact, and hope that they will help in the compilation by forwarding at once any changes in their addresses.

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TRANSACTIONS OF THE SOCIETY OF TROPICAL MEDICINE AND HYGIENE.

APRIL, 1917. VOLUME X. No. 6.

CONTENTS.

SPECIAL NOTICES.

1. The next Meeting of the Society will be held on Friday, the 15th June, at 5.30 p.m.

Tea will be provided for Fellows between 5 p.m. and 5.30 p.m.

2. The Secretaries hope to be able to issue a new Year Book this summer. They specially call the attention of Fellows to this fact, and hope that they will help in the compilation by forwarding at once any changes in their addresses.

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TRANSACTIONS OF THE SOCIETY OF TROPICAL MEDICINE AND HYGIENE.

JUNE, 1917. VOLUME X. NO. 7.

CONTENTS.

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TRANSACTIONS OF THE SOCIETY OF TROPICAL MEDICINE AND HYGIENE.

JULY, 1917. VOLUME X. NO. 8.

CONTENTS.

SPECIAL NOTICE.

The next Meeting of the Society will be held on Friday, 19th October, at 5 30 p.m.

Tea will be provided for Fellows between 5 p m. and 5.30 p.m.

EDITORIAL NOTICES.

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TRANSACTIONS

OF THE

SOCIETY OF TROPICAL MEDICINE AND HYGIENE.

NOVEMBER, 1916.

VOLUME X. No. 1.

Proceedings of a Meeting of the Society held on Friday, October 20th, 1916, at 5.30 p.m., at 11, Chandos Street, Cavendish Square, W., Surgeon-General Sir DAVID BRUCE, C.B., A.M.S. (*Vice-President*), in the Chair.

THE POSITION OF MALARIA IN SANITARY ADMINISTRATION.

BY ANGUS MACDONALD, M.D. (EDIN.), D.P.H., D.T.M.

International Health Commission, Grenada, West Indies.

The work of malaria administration and general mosquito elimination, instead of being relegated to Special Commissions with duplication of authority and indeterminate direction, must be recognised to be part of the routine work of the Health Department wherever that exists; and to take its place in due order of necessity, so far as finance permits, and the mortality and morbidity of the population demand.

In colonies where no health department exists; where a medical department retains the privileges and prestige of the age of cure and cannot conceive the meaning of prevention; or where an enlightened government and an enlightened medical department accept the preventive responsibilities without the necessary hygienic education or knowledge, sanitary improvement is a long way off.

In these the aid of commissions, with specialists *ab extra*, may accomplish something; but undoubtedly real advancement is only to be

secured where modern knowledge is placed at the disposal of a community in the form of an organised health department.

Executive responsibility under varying colonial conditions being retained by "Government" or placed on representative bodies, the first essential is a public health law, upon which may be founded the bye-laws which should deal with every item of sanitation that will come before the executive officers of the health department.

The staff of the health department in its simplest should consist of a Medical Officer of Health specially trained in practical sanitation, holding a degree in science or a diploma in public health, and of necessity in the tropics holding also a diploma in tropical medicine; sanitary inspectors, clerical staff, and labourers.

The number of Sanitary Inspectors depends on the population. For a unit—rural or urban—of 60,000 population, ten sanitary inspectors is a reasonable number. This population represents some 10,000 (urban) to 12,000 (rural) dwellings, and with ten inspectors it should be possible for each premises to be visited and reported on in routine once every three months, or four times a year.

I contend that the value of all public health administration is to be summed up in its domestic inspection and education; and, to get any control over that, the routine visitation is an absolute essential.

The crude notion that a sanitary inspector should go "nosing" around till he comes up against a nuisance is unfortunately, even at the present day, all too common.

Methodical inspection which reports in routine the normal as well as the unusual, the satisfactory as well as the nuisance, with its concurrent educational influence, will avail more than the vicious spy and prosecution system of administration.

The punitive sections of laws are to terrorise the wilful wrongdoer; wise public health laws and bye-laws teach the people what to do, and sanitary inspection is the personal education of the community in general. Wilful wrongdoing is rare; ignorance abounds; a fine of five shillings or a week in gaol will not bring sanitary surroundings to the poor and ignorant.

Of the ten sanitary inspectors the health officer will select his men for duties in which they display special aptitude. In general it will probably be found best simply to give them districts in which they have the entire

control of all sanitary measures, and to select men for special duty as occasion dictates.

A superior inspector may be chosen as a chief sanitary inspector, specially trained or not; but the health officer in any case, if he desires work done, will have to go down to his inspectors individually, go over their records with them, and systematically review their work in the field. *Sic itur ad astra*—only thus. If the health officer expects to control the disease conditions in a tropical community only by plotting curves and pulling strings at his office desk, he had better not attempt work in the tropics. He must see things done.

The Clerical Staff required to do the necessary work for the unit of population assumed will be two, a chief clerk and typist and a junior office boy. The due regulation of their work and apportionment of their duties the medical officer will adjust from time to time.

A Labouring Staff is inevitable in a tropical health department. In the city the scavenging rightly is an important section of the health department. The necessary carts, horses, mules, motor-vans, and all the paraphernalia of tropical scavenging will come under the direct purview of the health office. The manner of their economic engagement will be a matter for the interested authority. Their entire control during working hours will be under the medical officer.

Actual numbers for these need not be cited, conditions varying with population and area, with situation, and with class of labour and transport available.

On the labourer, strange as it may seem, depends the value of the ordinary antimalarial measures in an urban or rural tropical community. An average of a labourer per mile of streets, lanes, gullies and roads may be set down as a fair indication of the necessities of scavenging, street cleaning, gully training, gutter sweeping, and the various odds and ends of practical sanitation in a city. On the co-operation of the whole population depends the value of special antimosquito work, the suppression of Culex and Stegomyia, and sometimes of Anopheles.

In a purely rural community, outside towns, organised control of roadside gutters and other temporary breeding pools may be obtained by having a labourer roughly to five miles of roadway. This figure will vary enormously, owing to the great variety in condition of roads including first-class, secondary bye-ways, bridlepaths and footpaths which are under

authority; and, no matter under what different authorities they may be vested for constructive purposes, their sanitary control should be imposed on the health department. Only thus is there any hope of limitation of mosquito breeding.

The control of roads so far as the engineer is concerned is a matter of construction and æsthetics solely. Gangs of labourers are placed on the roads once or twice, or more often, in a year, to clean bush and train where anopheline breeding often has been going on for weeks or months before under no control; and, as soon as this display of zeal has passed along, the mosquito-breeding conditions begin behind.

The relation of the health department to the constructive department is one of calling on the authority to perform certain works. Here the health department may suggest a length of concrete gutter; there it may request training and levelling and the retention of the natural soil or subsoil drain. Again, it may request filling up, double channelling, and the like. Any or all of these suggestions may be accepted and acted on by the authority. But, accepted or not, it is still up to the health department to control the mosquito-breeding conditions, and prevent nuisance generally.

Then comes in the scheming of the staff: incessant watchfulness—here kerosene, there the brush, again the spade and rubble filling and so on.

Having postulated the details of the department necessary to tackle the whole of the problems of disease in a community, I shall now more particularly describe the special measures of antimalarial administration, including antimosquito measures generally.

These may be summed up as follows:—

1. Systematic Scrutiny of Death Returns.
2. Communication with General Medical Practitioners.
3. Routine Returns, from Institutions, of Malaria and other Mosquito-borne Diseases.
4. General Sanitary Measures.
5. Special Antimosquito Measures directed against:—
    i. Stegomyia in particular.
    ii. Culicines in general.
    iii. Anophelines.

I. SYSTEMATIC SCRUTINY OF DEATH RETURNS

is a substantial base for general sanitary operations. In respect of malaria, whose notification is not politic or practicable, the death returns give knowledge not otherwise readily obtainable, and facilitate the prompt investigation of the family history, residence and surroundings of the deceased. Careful investigation of the surroundings will justify the assumption of the accuracy or not of the cause of death; may reveal neglect on the part of householder or inspector, which allowed anopheline breeding where none should be; will indicate whether infection was local or imported; and the discreet reference to the certifying practitioner should pave the way for greater accuracy in certification.

II. COMMUNICATION WITH GENERAL MEDICAL PRACTITIONERS.

Friendly intercourse with his colleagues in general practice is of inestimable value to the medical officer of health. When, as I have stated above, the compulsory notification of malaria in a tropical community is not politic or practicable ("fever" being a commonplace of diagnosis and "malaria" too commonly the official synonym of "fever") the value of private information of the occurrence of malaria cannot be overlooked.

Concrete examples may aid comprehension of the importance assigned to this courtesy:—

> The health officer is called in consultation to a cottage where ten patients are down with malaria; several comatose. This cottage is within striking distance of a swamp whose reclamation is not feasible. By stocking with fish; kerosening edges, hoofmarks, wallows, etc., malaria had been banished from the area around and no case had been reported for some nine months. For several months kerosening had been intermitted on the ground of economy, and on the evidence of anopheline limitation. In the cottage were two visitors from a country parish, a notorious malaria haunt, who were "spending time" with their friends. The presumption is that they had brought the infection, which the probably clean local anophelines had promptly transferred to the entire family.
>
> Again, in town several cases of malaria are reported in a district where fruit gardening is carried on; irrigation is known to exist and is attempted to be kept under control. Blood examination

establishes the presence of the *Plasmodium falciparum* in several cases. Investigation shews laxity on the part of the Sanitary inspectors of this and an adjoining district. The nearest permanent anopheline habitat is two miles off, and yet at convenient points from these infected dwellings to the swamp, there are found stepping stones or flight pools harbouring, or capable of harbouring, larvæ, enabling anopheline communication to be maintained over the two miles.

Practical experience will multiply manifold these examples of the advantage to the health department of the sympathy and assistance of the general medical practitioner or other medical officers seeing patients.

III. ROUTINE RETURNS FROM INSTITUTIONS.

It should be possible to obtain from public institutions a return of all cases of malaria and other insect-borne diseases exactly in the same form as the notification of any compulsorily scheduled infectious disease.

Immediate investigation of such notified cases of malaria is of enormous value. In many tropical communities, too, it will mean practically all the cases that are found.

The facts elicited are :—

The residence and surroundings of the patient ;

The probable source of infection ;

The evidence of fresh infection or relapse ;

Whether malaria or not malaria ; and if malaria, the type of parasite ;

The seasonal and annual variation in number of cases ;

The age and sex incidence ;

The respective incidence and virulence of the different parasites.

The assistance towards control that is to be secured from the knowledge thus obtained, need only be mentioned to be appreciated by the average hygienist. None of the points need to be laboured ; but in regard to the actual parasite found and the comparative morbid results observed, a few words may be said, as they are points on which information is lacking and which ought to be obtainable.

There is certain evidence of the actual carriers of *P. falciparum*, *P. vivax*, and *P. malariæ ;* but in different localities different anophelines may be carriers of one and not of others. It might come to be possible

for the sanitary officer to say this or that case must have come from out side his district, as the mosquito carrier of that infection did not breed with him.

Again, there is not sufficient evidence of the morbific influence of the different parasites in spite of their colloquial differentiation into benign and malignant. Sufficient cases should be available in the accumulation of such records to establish the mass morbidity and mortality attributable to the different parasites.

IV. GENERAL SANITARY MEASURES.

It has been asserted above that there is no justification for divorcing malaria administration from the routine of the public health department. In the whole gamut, therefore, of sanitary measures there is general applicability, educational and executive, to malaria. So far as the health officer can get his laws and regulations to cover him, and so far as he may risk, failing these, he should treat the malaria case in general as he would any notifiable infectious disease, emphasising isolation, conducting disinfection, and enforcing rehabilitation of premises and general cleanliness.

There is always a moral and educative value behind much trifling and apparently useless disinfection proceedings.

Of course, the acute vulnerability of plague and yellow fever places them apart from other insect-borne diseases for general administrative purposes; and it is probable that control will be secured over malaria, filariasis, and certain other insect-borne diseases without the necessity to have them scheduled with and subject to the same statutory regulations as plague and yellow fever. But in these others the sanitary officer should go every step the law allows, and the little diplomatic step beyond, in his attempt to secure hygienic isolation (not seclusion) of those who are carriers of these diseases.

The chronic misery they inflict may well be more fatal to any community than the acute disaster of those more dreaded.

V. SPECIAL ANTIMOSQUITO MEASURES.

(a) Stegomyia fasciata.

The limitation of *Stegomyia fasciata* is a matter of incessant domiciliary visitation and education, and the results obtainable from rigid

inspection (without prosecution) are most striking. There are tropical cities at this moment probably harbouring stegomyia on 100 per cent. of premises, and it is open to a keen sanitary staff to reduce that record to 0·5 per cent. in a matter of twelve months.

A general sanitary scavenging clean up is a necessary preliminary, and ample scavenging and removal procedure must be maintained along with house-to-house visitation by capable inspectors, who will instruct householders in the law's demands and demonstrate to them the actual conditions of occurrence and means of prevention.

To detail the innumerable petty sanctuaries of *Stegomyia fasciata* need not be attempted: the creature is purely domestic, and will find breeding situations in any place or thing about buildings and yards that is capable of collecting a tiny drop of water. The wildest spot in which the writer has found *Stegomyia fasciata* larvæ was in rock holes by the sea, within one hundred yards of dwellings. He has also found them in a wild pine, on a tree near his residence, along with larvæ of *Wyeomyia pertinens*.

The sanitary inspector dealing with *Stegomyia fasciata* for any length of time will develop detective abilities of the highest order, but he will appreciate that with all his ingenuity he has still something to learn from it.

One or two examples of unexpected breeding places may be given to illustrate the domestic ubiquity of this insect:—

> The surroundings of an office habitually haunted with Stegomyia are repeatedly searched in vain, and it is almost concluded that the mosquitoes are coming from a distance. Accidentally larvæ are at last found in a small earthenware dish hanging on the wall, and dipped into daily by the copybook brush. Carelessly the dish for some time had not been emptied, but was filled up daily.
>
> In another case a few depressions in the tops of some pillars standing on a ruin were the source of mosquitoes troubling a neighbouring house. Again, after long search, it was found that a chance piling of timber under a heap of lumber caused the retention of rainwater sufficient to permit breeding long after drought had removed all ordinary possibility.

As has been said above the breeding spots of *Stegomyia fasciata* are innumerable; and from experience I believe it is safe to say they are

SWAMPY GROUND (LANTINI GRENADA)

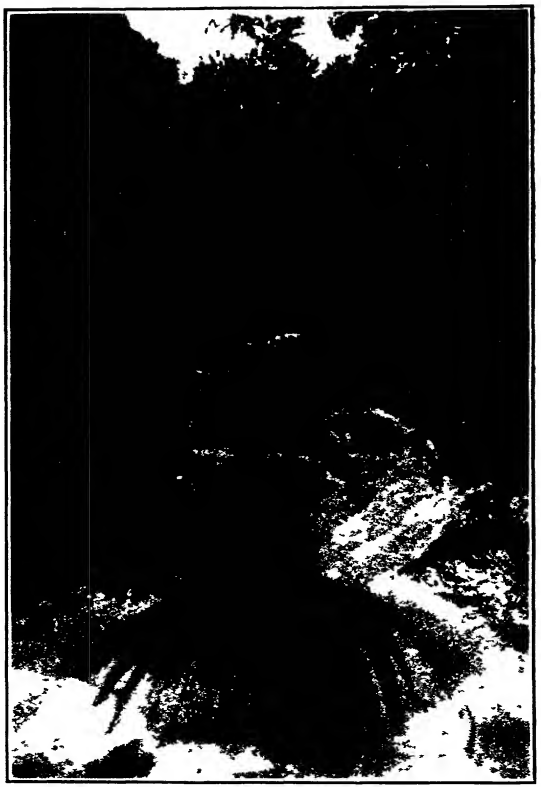
Left in moist condition after reclamation of swamp and probably now a far greater danger than before from Anopheline breeding. Note the Eucalyptus trees

LAGOON (GRENADA)

Swampy ground around contains Anopheline larvæ but the lagoon itself is harmless, and may be left as "a thing of beauty and haunt of game

BOULDER STREAM IN CEROA (GRENADA).

The local Anopheline does not breed here unless in tracts fully exposed to the sun



BOULDER STREAM IN CEROA (GRENADA).

The stream in flood should probably dispose of stray Anophelines.

IN A TROPICAL TOWN (ST. GEORGE, GRENADA).

A peep from a window – see cactus and various growth in gutter. Here the anomaly occurs of patients leaving the town for the country to recuperate from malaria !

GRENADA.

Moist Pasture, 800ft. elevation, on impervious rock, with abundant Anopheline larvæ practically the whole year round.

WAYSIDE DITCH (GRENADA)

Part concreted and only spasmodically tended Part beyond swarms with Anopheline larvæ

CONCRETING OF WAYSIDE GUTTERS

may create local safety against Anopheline breeding but too often the danger is merely handed on

SWAMPY GROUND (GRENADA)

Shaded by Cocoanut Plantation No Anopheline to be found although larvæ abound in the neighbouring wayside ditches exposed to the full glare of the sun

TROPICAL CITY (KINGSTON JAMAICA 1911)

An odd lot of tin cans and receptacles galore collected on one empty lot in a city with a Yellow Fever record

BY THE SEA (GRENADA)

Collecting *Stegomyia fasciata* larvæ from rockholes within one hundred yards of dwellings.

SWAMP (GRENADA).

Culexes in crabholes. No Anophelines.

always to be found within one hundred yards of where the mosquito rests.

(b) Culicinæ generally.

General culicine limitation—leaving out for the moment the consideration of extensive swamps and lagoons and estates whose agricultural necessities create and maintain mosquito breeding conditions—is a matter of the prevention by the householder of the existence of permanent or temporary collections of stagnant water, and of the suitable treatment of unavoidable pools, temporary and permanent.

It is the duty of the sanitary officer to see that the people understand the possibilities and dangers of the conditions, and that they are conversant with and obey the statutory requirements.

The sanitary officer will also have the preventive control of all conditions of water accumulation on public property, roadside gutters, gullies, etc.

The sanitary officer and governments must remember to have their zeal tempered by common sense, and to keep ever before them in all matters of mosquito elimination the questions, What are the mosquitoes before us? and what diseases do they cause? A frequent illogical sequitur, since insect-borne disease has been studied, is: Certain mosquitoes cause disease; therefore, all mosquitoes must be exterminated.

Acting in this spirit, sanitary officers have called on their governments, and perhaps in more cases the governments, from reasons of personal zeal, have taken the initiative without special advice, with the result that costly schemes of reclamation of lagoon and swamp; costly purchase of dredgers and pumping tackle; costly inexpert administration have been devised to the tune of thousands of pounds, when a few grains of expert common sense and a few pounds in labourers' wages might have disposed of all nocuous possibilities.

The actual dealing of sanitary administration on large estates and in general where conditions of irrigation and even of swamp are necessary from the agricultural standpoint, is a question for local and individual decision and compromise; and in all—cacao, banana, rice, cane, jute, rubber, etc.—means may be come to of safeguarding the lives of the workers to the economic advantage, and not the loss of the employers of labour.

(c) Anopheles.

Speaking generally it may be said that permanent anopheline haunts

in relation to eligible populations are few; while temporary anopheline haunts in touch with the mass of a people in inhabited country are many.

It follows that the first determination of the labours of the sanitary staff is toward the control of the temporary breeding places, which are many.

This is a point long insisted on and insufficiently realised. As a matter of fact its realisation is of little moment except in the possession of an organised sanitary department capable of acting on the realisation.

The control of malaria under widely varying conditions is one of the simplest of sanitary problems.

The mere limitation of Anopheles records results of almost magical appearance: notifications and deaths drop alarmingly! (I speak of the result solely of anopheline limitation).

Measures directed alone to the disease and the infected have little effect on the extent of the incidence of malaria on any community.

The antimalarial measures usually employed may briefly be detailed:—

1. Measures directed against Anopheles:—
   Insect trapping: Practically useless as a preventive measure.
   Larva killing by larvacides, chemicals and oiling, fish stocking, etc.; regular sweeping of channels, double channeling of watercourses with weekly deviation and consequent desiccation of larvæ: Of great value.
2. Measures directed to prevent mosquito breeding:—
   Regular bushing and training of channels, roadside ditches, etc.;
   Double channeling of watercourses with regular deviation;
   Regular sweeping of all water channels, gullies, gutters, ditches, temporary pools;
   Concreting of lengths of gullies, etc.;
   Rubble filling of stormholes, etc.;
   Grading of watercourses;
   Canalisation, with regular sweeping;
   Stocking of permanent pools, etc., with fish;
   Draining of land;
   Suitable cultivation to absorb swamp;
   Saltwater flooding of swamps and lagoons;

Reclamation of swamps and lagoons by opening up, clearing and bushing, filling, draining, etc.;

Cultivation of shade trees along the course of streams;

These all are of value and refer to temporary and permanent breeding places alike; one, or many, or all may have to be undertaken at one and the same time; and the important point to assure is that the law puts them all under the administrative control of the health officer.

To discuss these various measures in detail is needless; the individual sanitary officer must control their relative usefulness in definitive situations.

I wish, however, to emphasise again the importance of persistent control of the temporary as opposed to the permanent breeding places of Anopheles. The former are in contact with the people; the latter are not so, or readily may be avoided. Swamp has become a malaria fetish to the unskilled; and the demand is frequent for thousands of pounds to be expended on reclaiming a wide lagoon, when adjacent pastures, cultivations, and roadsides are the real danger, and whose control is a mere matter of pence to the authorities and property owners concerned.

Some inexpensive bushing and clearing and drainage around will often remove all danger that was supposed to come from swamp and lagoon; and these might be left as things of beauty and haunts of game. Even the Anopheles might be retained a harmless creature for the delectation of the future entomologist. For, as Ross has well demonstrated, limitation alone will rid communities of malaria; and so soon as intelligent preventive measures are instituted and rigidly maintained as a departmental routine, communities far and wide should be kept malaria free without any fabulous expenditure, the dread of which has retarded sanitary administration in the tropics.

3. Measures directed to the disease and the infected:—

These may be summed up in Quinine, Screening, and the Mosquito Net.

The mosquito net is a domestic necessity for the European in the tropics, but for the people at large its use is a practical impossibility.

Screening has its usefulness in certain situations.

Quinine has its position in relief and cure, but in sanitary administration it has none.

Continuous drugging with quinine may prevent attacks of malaria in anopheline countries, but statistics are still lacking to demonstrate the extent to which the taking of this drug prevents infection.

That the human individual is detrimentally affected by the continued absorption of quinine, be he infected with malaria or not, there can be little doubt. Whatever are the causes of blackwater fever, there seems to be a consensus of opinion that quinine has much to do with it—perhaps more to do with it than malaria has.

STEPHENS and STOTT, in the March, 1915, number of the *Liverpool Annals of Tropical Medicine*, record a study of the correlation between blackwater fever and quinine, and suggest lines of investigation demanding accurate figures, which it seems almost impossible to obtain in numbers sufficient to be of statistical value. I would suggest enquiry into the gross relativity between the quinine consumed in any community and the incidence of blackwater fever.

In the West Indies, blackwater fever is comparatively rare. More of it has been recorded perhaps from the Canal Zone. In malarial communities in Africa it is common. There seems to be a distinct connection between wide quinine taking and blackwater fever.

Statistical evidence—in my opinion of equal value to that desired by STEPHENS and STOTT, and more readily obtainable—might be furnished under some such enquiry as this :—

Country :
Population - *A*, Native and Creole :
B, White and Foreign :
Estimate of Malaria Incidence on *A* and *B* :
Estimate of Blackwater Incidence on *A* and *B* :
Estimate of Quinine habit (prescribed and self-taken) on—
A, Malarial :
Non-Malarial :
B, Malarial :
Non-Malarial :
Actual Quinine Import to country :
Quinine Consumption per head of population :

It might further be ascertained in most cases of blackwater fever whether any quinine idiosyncrasy was known independent of the presence of malaria.

I have said that *quinine has no position in sanitary administration;* and I *contend that the facile relief from malaria attacks by the taking of*

quinine, the consequent apotheosis of quinine in the hands of the medical officer and in the minds of the people, the easy glory of cheap quinine administration by governments, have disastrously postponed sanitary administration in the tropics, and prolonged personal misery and economic inefficiency.

Large masses of peoples without taking quinine recover from malaria between each seasonal infection. Quinine relieves suffering, and by destroying parasites limits the number of infective gametocytes. I search in vain for figures to demonstrate the eradication of malaria by quinine administration alone; but I find readily that whenever preventive organisation has operated, malaria disappears "like magic."

VI. EDUCATION OF THE PEOPLE.

This obvious necessity is nowadays sufficiently recognised that mere mention is almost needless. At the same time the right method of popular education is hard to come at.

In the writer's opinion the chief work of the medical officer of health should be educative—personally educative in the homes and assemblies of the people. That a well-salaried officer should find the detail of a medical or sanitary appointment in the tropics to be beneath his personal recognition, otherwise than in directing from an office, is unfortunately too common, and is almost an established prestige of Colonial medical appointments.

Administration which should centre in the lay offices of the government (with medical advisors) is placed on the chief medical officer; and the energies of an otherwise "good man" are prostituted to controlling financial and structural detail, repairing door handles of hospitals, and seeing that the out-porter carries a box for threepence instead of sixpence, while of medical or sanitary knowledge he soon ceases to have a care, knowing that his salary and pension are irrevocably unalterable and assured.

Where a medical officer attempts to keep up to date in scientific attainment, and assert for himself a position in his profession as surgeon, therapeutist, sanitarian, he finds that the professional aspect of his position clashes with the lay administration, and he either sticks to his profession and is dubbed "bad administrator," or he accepts the

easier alternative of forsaking his profession and becomes a "good administrator."

Lectures are valuable stimulants in popular education, the subject and style being determined to the class of people.

The lantern is a powerful adjuvant; and the cinematograph, where obtainable and suitable films are secured, is of value.

Leaflets are useful for any type of population; but in the poorer and illiterate homes the medical officer and staff should make it their duty to read and talk over the subjects of the leaflets.

The domiciliary visitation of the sanitary staff should be educative all along; people desire to be clean and to be healthy. Dirt is mainly a matter of ignorance. The efforts of mothers for the good of their children are so easily to be elicited by the friendly education of the medical officer and sanitary inspectors. Any medical officer who has realised and acted on the belief that his best hygienic results are to be got from domestic education, must have had frequent pictures of remodelled homes to smile upon him.

In respect of malaria, the breeding haunts may not always be associated with domesticity; but much of the personal prevention of malaria infection is a matter of "clean living"; and to say that the home is the fountain of sanitary progress is as true of malaria as of typhus.

VII. ENFORCING THE LAW.

I have little to say about this; if there were more education in the land, statute books would be smaller.

Wilful wrongdoing is rare; the villain of melodrama is not often seen in real life. When we seem to have found him let us remember "*Tout comprendre c'est tout pardonner*," and realise that education rather than law is demanded; that in sanitary progress law too often spells stagnation, while education spells health.

To sum up the special references to malaria, I ask for the realisation that:—

The care of the health of tropical communities demands the general establishment of a Preventive Medicine Department apart from or part with existing medical departments, but, in any case, with specialised and experienced heads;

Malaria administration, to be of most advantage, should be part of the routine of any health department;

Malaria, in general, is acquired seasonally more from temporary than from permanent breeding places of anophelines; control of these temporary danger spots is a simple and inexpensive matter in the presence of the postulated health department;

Quinine administration for the relief and cure of malaria may be left to the medical practitioners, and should form no part of the armamentarium of the sanitary department.

In conclusion, I owe it to say that my opinions are founded on direct observation of malaria and malarial conditions in the West Indies and Central America; and certain animadversions may, to some, appear ridiculous when applied to other situations.

General study, however, of the literature of malaria and of the Reports of the Colonies leads me to believe that the main propositions hold universally, and that adaptation locally is the duty of the responsible officer.

Discussion.

Professor W. J. Simpson: I would like, first of all, to congratulate Dr. Angus Macdonald on his views—and robust views too—which he has expressed. He has very wisely limited himself to the conditions that he knows in the West Indies. He makes a surmise with regard to the medical reports of other colonies which he has read, and I may say that his surmise is correct. I am in entire agreement with him that malarial administration and the extermination of the mosquito should be in the hands of the Health Department as a matter of routine.

I assume that when he speaks about special commissions he is not referring to Scientific Commissions, but to Commissions which perhaps undertake work which can very well be left to the Health Department. Since the monumental discovery made by Sir Ronald Ross that malaria was a mosquito-borne disease, it then became the duty, I think, of the Health Department to apply that knowledge to the prevention of malaria, just in the same way as it deals with small-pox, cholera and typhoid fever. It is a matter of routine for the Health Department to remove the conditions that are favourable to

the causation of such diseases, and accordingly, in my opinion, it should be the duty of this Department to remove the breeding places of mosquito larvæ. It has been shewn by Dr. MACDONALD that these measures are not expensive, as a rule, if they are done in a systematic way. I quite agree with him that there is far too much inclination—in the popular mind—to fill up swamps and to undertake large works. Of course, there are certain swamps which must be filled up; but my experience agrees with that of Dr. MACDONALD that most of the malaria is due to the mosquitoes' breeding-places being close to dwellings. If the dwellings are quite close to the swamp, that is a different matter. There is also too great a tendency on the part of the Government to have water-tight compartments for different diseases and their prevention. I think it is a wrong policy. I believe that when special Commissions have thrown light on the causation of any particular disease and the methods by which it can be combated, the duty of dealing preventively with the disease should at once become the duty of the Health Department. But, at the same time, there must, with the increase of duty, be provided the staff for carrying out the extra work. As I have said before, I am assuming that Dr. MACDONALD'S references are not to Scientific Commissions. Nor, I should say, would they refer to the devising of new organisations for Health Departments; nor for consultation with sanitary experts, men who have perhaps devoted their lives to special subjects. I take it that the Health Officer in a far-off land is very glad to have the expert advice of a consultative scientific Sanitary Officer, in the same way that the practitioner at home is, at times, glad to have a consulting physician or surgeon to give his opinion on a case or subject.

Special commissions are also necessary for studying the cause of some new disease, and determining the methods to prevent it. But when these are known the application should certainly be held to belong to the Health Department.

With regard to other Commissions, it seems to me that there is a danger of overlapping, and a further danger that when the Commission goes home no further progress is made. I might give an illustration, such as the International Commission for the Prevention of Ankylostomiasis. I fear that if this does not fit in with the Health Department, as soon as that Commission goes away, and there is no provision for the Health Department to carry on its work, no further progress will

be made. Similarly, there have been a number of Malarial Commissions in India, which have concerned themselves mostly with indexing certain mosquitoes and their habits. But I hold the view that since Ross's discovery it would have been much more useful and more practical if methods had been devised for creating and organising a Health Service in villages and towns, and putting that discovery into practice, which can only be done by a proper Health Service.

I agree with Dr. MACDONALD that there can be very little progress without health laws. These health laws are the embodiment of the practical knowledge that we have gained concerning the improvement of the health condition of communities, and it is most advisable to have these health laws. But I am a little sceptical of the view which he expresses that health laws can be effectually put into operation by persuasion. He speaks about mothers being anxious to carry out the cleanliness of the domestic dwelling, and so on; but there are very many cities, especially in the East, where there are but few such mothers to be found, there being in those parts a large migratory population, and there the only "persuasion" is the enforcement of the law, with heavy penalties for failure. I believe under these circumstances that enforcement of the law is the very best teaching, even far better than lectures. It makes an appeal to the pockets of the individuals, and they soon learn that it is rather expensive to break these laws. There are many places that have no health laws and no health administration. Then there are places with health laws but no Health Department. There are also places with a Health Department but without good health laws; and there are places with a Health Department and health laws and yet the laws are not enforced. One has seen excellent health laws on the statute book which are not enforced until an epidemic occurs, and then only during the epidemic.

There is one point which, I think, has been omitted from the organisation which Dr. MACDONALD speaks of, namely, the Health Laboratory. I consider that any town of the dimensions he spoke of, or even a smaller town with a rural area around it, ought to have a Health Laboratory. It is there that important information is very often obtained about diseases. I remember that when I first went to India a laboratory was one of the first things I had established after the organisation of the Health Department had been completed in regard to the scavenging, the inspection and

so forth. I found that the laboratory helped one immensely in dealing with cholera and other diseases.

There was an interesting subject which I came across when I was in Dar es Salaam. Professor KOCH had introduced a system there of giving quinine to the people in order to reduce the malaria. I had a conversation with Dr. ORENSTEIN, who was the Medical Officer of Health there at the time of my visit, and he had been Colonel GORGAS' assistant at the Panama Canal. He found that after a time the treatment of malaria by quinine was given up, not having been the success that Professor KOCH expected. And Dr. ORENSTEIN had come to the conclusion that the best methods for adoption were the elimination of the mosquito and the draining of certain swamps which were close to dwellings.

I certainly think that we are very much indebted to Dr. MACDONALD for his paper, and the conditions he has shewn us to exist in Trinidad.

Mr. T. P. BEDDOES: There is no doubt that a great deal has been done in Trinidad, and one extremely appreciates the way in which the efforts have been detailed to us. But one cannot help seeing that, not only there but in most of the West Indian Islands, and everywhere under the control of the Colonial Office, there is not that enterprise and that harmony of work that there should be.

In the first place, there is the question—dealt with here and referred to in so many places—that the medical officer is simply a servant, to do just what he is told; and the officials sit down and say the Medical Officer will do what he is told. And if he suggests that they should apply their minds to aid in determining the best way of getting rid of the anopheles, we find, possibly, that some man fills up a big pond or lagoon, which is of no use whatever. Then it is said that so much has been spent on that that there is no more money available. But the real fact is that there has been an unthinking amount of enterprise which has been limited simply by the amount of cash, it is not a question of the really useful work which has been done. There is no doubt that is the tendency of human nature. And, if anything, we see that more frequently now than at any other time. We find even in the London daily papers comments to the effect that members of the profession are to do what they are told, or else the public will think they are ignoring the welfare of the children and the nation.

When we come to the case of the small islands of the West Indies we see the need for officials to be energetic and to work in harmony; and particularly those abroad must have sympathetic support from the officials at home, and of that, up to now, there has been great need, and still greater lack. Individual efforts, such as those which have been put before us to-day, are of limited utility, because in one island one thing happens, and in another island another thing is done. There is no doubt that the real welfare of the tropics depends on health, and at no time in the history of the world was there such a shortage of food as there is now. Yet it is certain that food can be raised more economically, can be transported into the large towns more economically there, than in any other part of the world. There is sea transport, which is the cheapest form of transport; and instead of bringing it a long way over land, as sugar is often brought from Russia, and from another part which I need not mention, England particularly, and Europe generally, could have a much more plentiful food supply if efforts such as we have been told of to-day were really and properly organised, and received due help from London.

The CHAIRMAN (Sir DAVID BRUCE): We must thank Dr. ANGUS MACDONALD very heartily for his interesting and very useful paper.

He said in one part of his paper that "continuous drugging with quinine may prevent attacks of malaria in anopheline countries, but statistics are still lacking to demonstrate the extent to which the taking of this drug prevents infection." It is evident from that that Dr. MACDONALD is not a great believer in the efficacy of quinine as a prophylactic. I have often wished that this matter could be settled once for all by good evidence, but that has not, as far as I am aware, been done. I remember taking a party of natives, twenty years ago, into a very malarious part of Zululand. At that time the agency of the mosquito was still in the position of an hypothesis, but quinine was considered to be a protective against malaria. I therefore gave the natives ten grains a day, and took the same quantity myself. The tent was put up at the side of a swamp which was solid with mosquitoes, and in spite of the quinine we all got malaria. Ever since that time I have had a sceptical feeling in regard to the use of quinine as a prophylactic against this disease, and, as a result, I have never taken it as a preventive when in a malarious

country. Many white people when in malarious countries take five or more grains every day as a matter of routine, and continue the practice for years. If this could be definitely proved to be useless, it would save a deal of discomfort.

Dr. ANGUS MACDONARD: I thank you, sir, and you, ladies and gentlemen, for your appreciation of my paper, and I would thank Professor SIMPSON especially for his evident personal sympathy with my points of view.

As regards commissions, Professor SIMPSON is right in assuming that in my paper I did not stigmatise Scientific Commissions; what I spoke of was the type of local Commission which is composed of clergymen and ladies who have but little else to do. I referred to the sort of lay Commission which, in some colonies, is given the absolute administration of antimalarial measures, utterly regardless of the existence of any Sanitary Department whatever. And even our special commissions have their dangers, and, as Professor SIMPSON admitted, they are not always followed up with the practical work that the knowledge obtained, and demonstrated to the point of proof, demands. Even in reading the Reports of the Yellow Fever Commission one is disappointed at seeing whole volumes detailing investigations, when we must know that for many years, ten at least, the cause—whatever the ultimate particle of infection—the stegomyia, has been there. And alongside of these reports of the investigations in West Africa there is obviously an undercurrent running through it all suggesting that there is little or no practical work going on. The mere limitation of stegomyia seems to be absolutely neglected, as was evidenced in the same Report of the Commission. That emphasises what Professor SIMPSON said as to the Reports of these Commissions being acted upon too late.

With regard to the International Health Commission, that is a name for a body of American gentlemen who have been given the control of some millions of JOHN D. ROCKFELLER'S wealth to carry out one definite purpose, and that is to put the money into any part of the world, including those British Colonies which desire to accept it, and simply to ascertain who are infected with ankylostomiasis, to cure them, and to get as copious figures as to cures as they can. That offer has been accepted by our Colonies in many instances outside any Medical or Sanitary Depart-

ment. What the ultimate end or accomplishment of that Commission may be it is hard to see just now, because war events have complicated matters in the direction of this work having become an American affair. It has prevented our own men being set aside for that work, so that American medical men are at work in most of our Colonies, instead of, as in normal times, British medical men in British Colonies.

I realise and quite appreciate Professor SIMPSON'S point that my statement about persuasion in sanitary matters cannot be taken absolutely. There are situations in which circumstances may differ widely, and in some the firm hand of the law must be used.

I grant at once that my neglect to mention a Health Laboratory was an unfortunate omission, because that is an absolute essential. As for myself, when doing practical public health work in the West Indies, I made my own little laboratory, and in that way did what I could; and in Jamaica, at any rate, I had the help of the Government Laboratory for anything I could not undertake myself.

I cannot say much concerning Mr. BEDDOES' remarks, except that I believe he has a sympathetic feeling as to the difficulties which men anxious to do work in the West Indies come up against. That practically touches on the methods of administration, which have not, in my opinion, kept pace with the times, but which present events may quickly modify.

Sir DAVID BRUCE cheers me by his direct evidence of the non-prophylactic effect of quinine. Possibly he may in time become so sceptical about it as to be entirely with me.

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THE DEVELOPMENT OF PATHOGENIC PROPERTIES IN PROTOZOA, WITH SPECIAL REFERENCE TO THE HERPETOMONAD GROUP.

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Recent advances in our knowledge of the developmental cycles and incidence of certain protozoa which cause disease in man and vertebrates, enable us to speculate with some justification as to the laws which govern and the factors which induce the appearance of pathogenic and lethal properties in unicellular micro-organisms.

It may not be out of place to remark that the existence of protozoa harmful to man and the bigger mammals has focussed attention on the whole phylum, and stimulated investigation and research with very satisfactory results. We are, therefore, not only justified in considering

and studying the developmental morphology of any single group of protozoa, but also in observing the general biological rules which can be deduced from the behaviour of various protozoa under different circumstances, and comparing them with the conduct of their affinities.

Such discussions have been described as irrelevant, futile and unprofitable. They certainly do not always deserve this severe stricture. Anyone interested in research in the tropics knows the feeling of insecurity when observing apparently unknown or exceptional facts, which results from the knowledge that it is not possible to submit the result of one's fleeting observations to the crucial test of expert criticism—the surest and best incentive to research. Progress follows both inductive and deductive methods; it is not advisable to use either alone to the exclusion of the other.

It is quite comprehensible that the subject-matter of this paper is closely linked up with the question of parasitism, because though "parasitis" does not necessarily mean harmful or "pathogenic," yet it is evident that pathogenic properties cannot be evolved or become noticeable without a more or less prolonged stage of parasitism.

Parasites have been often classified and tabulated, and the terms employed for the purpose are generally known, *e.g.*, permanent parasite temporary parasite, obligatory parasite, facultative parasite, erratic parasite, etc. As a rule, only a small proportion of the terms employed in this connection are applicable to protozoa.

The views expressed in this paper are based on the behaviour of certain protozoa in their relation to man and other vertebrates. An attempt has here been made to classify various unicellular microorganisms according to their biological features, whilst zoologists and protozoologists group the objects of their study according to some permanent or transitory morphological characteristic.

It is also evident that the term pathogenic applied to any protozoon designates the properties of some phase of its cycle in relation to some animal or group of animals; in fact, the word "pathogenic" describes the sum of the reactions between host and parasite.

I have ventured to tabulate protozoa of medical interest in the following fashion:—

Free-living or sapropelic. Ex., Infusoria.

Epizoic: all pathogenic, some even lethal. Ex., Ichthiophthirius, Leishmania.

Entozoic
- Saprozoic, usually enterozoic. Harmless, possibly even beneficial. Ex., Chlamidophrys.
- Pathogenic
  - Lethal. Ex., *Trypanosoma rhodesiense.*
  - Non-lethal. Ex., *Leishmania tropica.*

By considering the various fashions in which they damage their hosts we may divide protozoal parasites into those which destroy or necrose, such as *Entamœba histolytica*, or are proliferative, such as Rhinosporidium; or others which extract and possibly convert to their own use some necessary body substance, such as is done by *Plasmodium malariæ* with hæmoglobin. Or the damage may be purely mechanical, as we see in Sarcocystis. The lethal properties of certain trypanosomes do not appear to be due to toxines which can be demonstrated in the usual fashion; yet a "diffusive" pathogenic action is to be expected from the group.

If, again, we consider the localisation of the protozoa in their principal (vertebrate) host we see that the following variations of parasitism have been observed:—

Intranuclear = *Cyclospora caryolytica.*

Intracellular = *Plasmodium malariæ.*

Plasma parasites = Trypanosomes.

Intestinal = Giardia (Lamblia).

Intercellular = Sarcocystis.

In addition, I should like to make a distinction between a host that harbours a harmless parasite and one that is preyed upon by a pathogenic protozoon; the former is simply a host, the latter a victim.

The nomenclature having been settled, we can now proceed to consider and compare the properties of various pathogenic protozoa, including spirochætes and their relationship to free-living and saprozoic forms.

It is true that spirochætes are not protozoa, and DOBELL has ably pleaded their classification among bacteria. It cannot, however, be denied that at times they behave like protozoa, or, to sum up the situation according to FANTHAM, "that spirochætes are intermediate in character between bacteria and protozoa, shewing morphological affinities with bacteria, and physiological and therapeutical (biological) affinities to

protozoa." The following are the reasons which seem to place them in a group of "Proflagellata," intermediate between Protophyta and Protozoa :—

Bacterial characteristics.	*Protozoal features.*
1. Morphology very similar to that of certain known bacteria.	1. Longitudinal division.
2. Absence of definite nucleus.	2. Difficulty of artificial culture.
3. Transverse division.	3. Terminal, single flagellum.
4. Granules similar to spores.	4. Transmission by arthropods.
	5. Influenced by arsenical drugs.
	6. Movements follow a certain type of progression, whilst bacteria career aimlessly across the field of vision.

The argument of immunity is not decisive on either side, because agglutinating sera can be demonstrated against spirochætes, which though clumping in a higher dilution than those which are known against trypanosomes, yet are feebler than those which can be got by the injection of true spirilla.

In reviewing the principal features and properties of protozoa capable of causing disease in man and animals we note the following points :—

TREPONEMATA.

1. Free-living : saprophytic and pathogenic races.

2. Several pathogenic varieties. Habitat : plasma, inter- and intracellular.

3. Close resemblance between saprophytic and pathogenic races.

4. Hosts : numerous vertebrates. Victims : birds, mammals.

5. Transmission to victims : arthropods, especially ticks and lice. Also, by direct contact, *T. pallida* and *T. pertenue.*

HERPETOMONADS.

1. Free-living : saprozoic and pathogenic races.

2. Several pathogenic varieties. Habitat : epizoic and entozoic, intracellular.

3. Close resemblance between all varieties.

4. Hosts : arthropods, reptiles. Victims : mammals.

5. Transmission = ?

TRYPANOSOMES.

1. No free-living species, but varying degrees of pathogenicity.
2. Numerous more or less pathogenic species. Habitat: plasma.
3. Lethal species usually. Morphologically distinguishable from non-pathogenic.
4. Hosts (?) and victims: all classes of vertebrates.
5. Transmission: Hirudinæ, blood-sucking arthropods, or direct contact *(T. equiperdum)*.

AMŒBÆ.

1. Free-living: saprozoic (enterozoic) and pathogenic races.
2. Pathogenic varieties not numerous. Habitat: enterozoic and intercellular.
3. Pathogenic (lethal) species, similar to but distinguishable from simple commensals.
4. Hosts: practically every animal with a digestive tract. Victim: man.
5. Transmission: ingestive.

PLASMODIDIÆ.

1. Free-living: unknown. Affinities with Coccidia.
2. Numerous pathogenic varieties. Habitat: Erythrocytes.
3. Common features between all pathogenic stages; morphological differences between stages seen in birds and mammals.
4. Hosts: arthropods. Victims: birds, reptiles, mammals.
5. Transmission: arthropods. (Blood-sucking diptera in birds and mammals).

BABESIA.

1. All pathogenic, some lethal.
2. Numerous varieties. Habitat: Erythrocytes.
3. Considerable morphological resemblance between various species.
4. Victims: mammals.
5. Transmission: arthropods (ticks).

The purpose of this necessarily incomplete classification is to shew that the parasitic protozoa which have the most marked pathogenic properties for man and mammals are those which, as a rule, can be definitely distinguished morphologically from their saprozoic and harmless

relations. In other words, that a complete adaptation to a parasitic mode of life, with its corresponding alterations in morphology, results in an attendant enhancement of virulence. We must exclude from this rule the Herpetomonad group. The pathogenic, even lethal properties of *Leishmania donovani*, and the extreme similarity of its herpetomonad stage to saprozoic races, are a definite and glaring exception to this rule, which requires separate attention and explanation.

What does the increase in virulence consist of? It does not appear to be possible to frame any law which will adapt itself to all cases. We have already seen that the intensity of pathogenic action of single, even related protozoa varies within wide boundaries. One feature, however, appears common to most pathogenic protozoa—the facility and rapidity with which they multiply in their victim's body after a more or less prolonged period of incubation has been overcome. This might be explained by the necessity of maintaining the species in face of the evident disadvantage of this protective measure, which hastens the dissolution of the victim and menaces the parasite with extinction. It is an evident paradox that most lethal protozoa appear to be bound to their victims and an intermediate host; they are incapable of a free life-cycle, and, therefore, must at all costs gain entrance into their intermediate host before their victim dies, or else meet the same fate.

It is quite evident that a lethally pathogenic parasitism is not in the interests of the individual victim, and also not in the interest of the parasite. Protective measures exist to a certain extent in single instances, such as encystment in amœbæ and the presence of "reservoirs" in connection with pathogenic trypanosomes, such as *T. rhodesiense* and *T. gambiense*.

As to the trypanosome group we can see that the most virulent and pathogenic flagellates, though they are easily transmitted by inoculation from one animal to another, yet can only be cultivated with extreme difficulty if at all; some of the cultivations which have been published are merely conservation of trypanosomes in a suitable medium. On the other hand certain slightly pathogenic trypanosomes, of which we may take the *T. lewisi* as an example, are easily cultivable in artificial media, where they transform into crithidial and herpetomonad shapes.

Comparing these observations together, the conclusion can be drawn

that in the case of the trypanosomes of great virulence, such as those which have been mentioned, also *T. evansi*, *brucei*, etc., the adaptation to a certain form of parasitic life has become so permanent that a reversion to simpler forms is no longer possible. On the other hand, *T. lewisi*, with all its mild pathogenic properties adapts itself easily to varying conditions, and in so doing repeats its ontogenetical development in a somewhat similar fashion to what has been observed in the embryonal stages of vertebrate life.

These views, however, break down completely when dealing with herpetomonads, for though the leishmania stage is nothing more than an encystment, which can be easily brought about by injecting the flagellate in artificial culture into the peritoneum of a rat or mouse. It is true that this encysted stage can multiply without returning to its flagellate form, but still it can be turned into a flagellate with the greatest ease by placing some of the virus in citrated saline. Therefore we are face to face with a protozoon which is distinctly pathogenic, and yet has not adapted itself in a permanent fashion to parasitic life, because its morphological features are capable of reverting to those of free-living and saprozoic types.

In this connection it is well to consider the experiments of FANTHAM and PORTER, which shed much light on the subject. The conclusions of these two workers deserve to be quoted *in extenso* :—

> . . . . . researches have been conducted on the introduction into vertebrates of flagellates normally parasitic in insects. The vertebrates became infected by inoculation with the flagellates or by being fed on the insects containing the protozoa. . . . . Flagellates from sanguivorous and non-sanguivorous insects were used, and cold-blooded as well as warm-blooded vertebrates as hosts. The introduced protozoa were pathogenic to the mammals, but not markedly so to the cold-blooded vertebrates. *Herpetomonas jaculum*, *H. stratiomyiæ*, *H. pediculi*, and *Crithidia gerridis* (parasitic in certain water-bugs) proved pathogenic to mice. A puppy was infected by way of the digestive tract with *H. ctenocephali*. Frogs became infected with *H. jaculum*, lizards with *C. gerridis*, and sticklebacks with *H. jaculum*. Second and third passages of some of the parasites were obtained. The protozoa, whether *Herpetomonas* or *Crithidia*, were present in the vertebrate hosts in either the non-flagellate or the flagellate form, or

usually both. They were most abundant in the internal organ of the hosts, more particularly in the liver, spleen, and bone-marrow.

* * * * * *

It is inferred that the various leishmaniases are due to a herpetomonad of invertebrates which, under different conditions of environment, produce pathogenic effects in very varying degrees in different vertebrates, from zero, as in the mice described by DUTTON and TODD in 1903, to high mortality, as in Indian kala-azar, and probably zero again in cold-blooded hosts. It is also a flagellate which can probably live in invertebrates not already recorded as being infected. A human reservoir of leishmaniasis may occur in some places, while warm- and cold-blooded vertebrates may also function as the same. (FANTHAM, l.c. page 346).

Some years ago I described a herpetomonad found in the cloaca of *Chamæleon pumilus* at Robben Island. Its description has been published in 1913, and at that time I remarked that its principal interest consisted in the fact that it appeared to be a transitional stage in the development of herpetomonads from saprozoic protozoa in the gut of insects to permanent parasites in the cloaca of a vertebrate. Since then further specimens have been sent me by my late assistant at Robben Island, Mr. J. CAMPBELL, so, that the occurrence of this interesting protozoon in the cloaca of the South African Dwarf Chameleon does not appear to be a rarity.

If we now consider the results of FANTHAM and PORTER'S experiments and the appearance of the herpetomonads found as saprozoits in reptiles, in the light of the experience that a pathogenic protozoon in a vertebrate is usually morphologically distinguishable from its harmless relations, we may be allowed to venture the suggestion that the anomalous position taken up by the Leishmania group is either due to the fact that their pathogenic parasitic and disease-producing rôle is a recent acquisition ; in fact, if we consider that notwithstanding diligent search up to the present, no acknowledged intermediate arthropod has been found for oriental sore or kala-azar, we may come to the conclusion that it is quite possible that this group of diseases is acquired by the inoculation through blood-sucking insects of apparently free-living or saprozoic varieties.

The deductions of FANTHAM and PORTER's work would therefore lead to a logical conclusion.

Reverting now to the behaviour of pathogenic treponemata, we see that according to the views expressed above, it appears that the striking morphological resemblance, amounting to morphological identity, which is found between *Spirochæta dentium* and *Treponema pallida*, which otherwise differ so greatly in their harmful properties, is certainly more like to what is observed among bacteria than protozoa. Among bacteria we have at times the greatest difficulty in distinguishing virulent from non-virulent strains; among protozoa, with the possible exception of certain infusorians of the Balantidium class, we have no such unsurmountable difficulty, especially if the anomalous behaviour of herpetomonads can be explained.

Arising out of these suggestions, renewed attention can be paid to the question of the origin of disease-producing protozoa. It is difficult at the present moment to bring forward any strikingly new views on the subject; still, a plea can be made for a broader view of the problem. A single explanation will hardly suit all instances which differ in so many particulars. In the case of *Entamœba histolytica*, simple repeated ingestion of cysts will give a satisfactory answer. For the Leishmania group the contamination of the wound, produced by a blood-sucking arthropod, with the contents of its gut, may possibly be applicable as a theory regarding the first steps towards parasitism in vertebrates. Pathogenic trypanosomes are possibly definitely specialised descendants of the harmless variety, which again are related to herpetomonads. The big group of Plasmodia present, however, quite a different riddle for solution. They are evidently permanent parasites of arthropods, or at least they pass the greatest and most important part of their life-cycle in arthropods, as real intercellular parasites, not simple inhabitants of the intestine. The adaptation to parasitism in vertebrates must therefore have taken place through regurgitation or direct inoculation through an insect sting. Three alternatives are then at least available to explain the initial acquisition of parasitism and disease-producing properties in protozoa. In course of adaptation to a parasitic mode of life other and new peculiarities were acquired, in addition to parasitism, as a result of the facility in obtaining nourishment from the host or victim; in addition

the higher temperature found in warm-blooded animals would encourage rapid division and multiplication, and therefore tend to perpetrate all the more any acquired morphological peculiarities or distinctive features.

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DISCUSSION.

Sir PATRICK MANSON (answering the invitation of the Chairman): I have no great desire to open the discussion on this paper, because the subject is far too complicated: it requires considerable thought before one could hope to evolve anything like a hypothesis on these matters. I think a hypothesis is a good thing, and, of course, it may be either right or wrong. And Dr. BAYON'S hypothesis may prove to be correct. Meanwhile, I think, it is better to suspend judgment. Perhaps someone more competent to open the discussion will do so.

A NOTE ON THE EGGS OF THE LIVER FLUKE, *CLONORCHIS SINENSIS*, VAR. MINOR (VERDUN AND BRUYANT), 1908.

BY

C. H. TREADGOLD, M.D., CAPT. R.A.M.C.

Recently, while examining fæcal material for amœbic cysts, I came across the eggs of this parasite in an Australian. The patient had visited different parts of China, and had also lived in places in Australia where the vegetables were exclusively supplied by the Chinese. The question as to which country was responsible for his infection is interesting in view of the fact that this trematode is held to be the cause of a serious disease of the liver which may terminate fatally; and in view of the fact that, up to the present, its eggs do not seem to have been reported in Australians who have never left their country. Unfortunately, our knowledge of the life history of these parasites is very limited. The only experimental work I can find on the subject was done by KOBAYASHI,[1] who claimed to have infected cats, rabbits and guinea-pigs with the cysts contained in the uncooked muscles of different fresh-water fish. Still, other sources of infection may exist, and in the absence of proof to the contrary we must admit the possibility of encysted cercariæ being ingested with imperfectly washed salad, grown in water contaminated by the excreta of infected orientals.

The Morphology of the Eggs.—The smallest seen measured 20·8 by 13μ, the largest, 29 by 16μ. The lid of the shell was rather flat, its margin being but slightly indicated; in fact, the operculum was only definitely made out in the minority of cases. The constriction described as existing

towards the anterior end was seldom well defined, but a small projection was occasionally noticed at the posterior extremity. It will be seen that the eggs differed appreciably from one another, both in size and appearance, and the question at once arises as to whether they all belonged to the same species. To decide this question a brief reference to the literature of the subject is necessary.

In 1883, BAELZ[2] gave a description of two new trematodes—common enough in Japan—*D. endemicum* and *D. innocuum*, the former alone being pathogenic. He described the eggs as varying between 20 and 30μ in length by 15 and 17μ in breadth, with a lid at the narrowest pole and frequently a projection at the other end.

In 1886, BLANCHARD[3] grouped the two forms described by BAELZ under the heading *D. japonicum*, regarding the differences as being too slight to justify the creation of separate species. BLANCHARD[4] states that the eggs vary between 23 and 30μ in length by 13 to 16μ in breadth, but that earlier observers give from 20 to 36μ in length by 15 to 20μ in breadth.

In 1907, LOOSS[5] described these parasites under the names *Clonorchis endemicus* and *Clonorchis sinensis*, the former being pathogenic and met with chiefly in Japan, the latter feebly or non-pathogenic and found principally in China. According to this observer the eggs of *C. endemicus* vary from 26 to 30μ in length by 13 to 16μ in breadth, while those of *C. sinensis*, although they do not differ markedly in length, are slightly wider (15 to 17μ). The narrowing towards the anterior end in *C. endemicus* is, in the main, not so marked, and the margin of the rather flat lid is not so sharply projecting as in *C. sinensis*; but these differences are, on the whole, very slight and not recognisable in every specimen.

VERDUN and BRUYANT[6] state that both styles of egg, as described by LOOSS, may occur in the same specimen. They regard the validity of the species *C. endemicus* as being insufficiently established, and think it more logical to create two varieties, *C. sinensis major* and *C. sinensis minor*, the former found chiefly in China, the latter—the pathogenic one—chiefly in Japan, Tonkin and Annam.

Conclusions.—The eggs found in this Australian patient correspond

approximately with the description given by Looss for *C. endemicus* (*C. sinensis minor* of VERDUN and BRUYANT).

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DISCUSSION.

Dr. R. T. LEIPER: I do not know that there is much that I can add to this interesting paper. Attention may be drawn perhaps to a recent publication, in which KOBAYASHI shews that the measurements of the eggs given by LOOSS as characteristic of the different species of clonorchis are found in different specimens of a batch of worms reared experimentally by infecting cats from certain freshwater fish. He also points out that the only specific difference given by Looss—the presence of a certain amount of pigmentation in *C. sinensis* in contrast to that of *C. endemicus*—is not a true specific difference, but occurs also in specimens reared in his experiments. The third point on which Looss differentiated the two species was the size. He has made out that *C. sinensis* was a much larger parasite than *C. endemicus*. But I think KOBAYASHI has pointed out that this also is incorrect. It should be noted, however, that when Looss wrote his paper he did not know that freshwater fish were transmitters of the disease. Now, a person eating an infected fish would probably take in several hundreds of the encysted cercariæ at one time, and probably these would grow fairly uniformly.

So that if a *post-mortem* examination were done some months later, most of the parasites would be found to be of the same size. This will perhaps explain why, in batches of material sent by collectors, there has been that remarkable uniformity of size, which has given rise to the notion that there are two species or varieties in *C. sinensis*. Of course, the fish is only the final intermediate host. Like all digenetic trematodes there is a mollusc involved as well. The essential larval metamorphosis is suspected to take place in a species of Melania. The terminal stage passes into the fish, which merely acts as a passive vehicle for the encysted cercariæ to the final host.

The author says, "We must admit the possibility of encysted cercariæ being ingested with imperfectly washed salad, grown in water contaminated by the excreta of infected orientals." I think we can only admit that if the encysted cercariæ have passed out of the fish into the water, and so contaminated the salad. That is a possibility, but, I think, a very remote one.

In the case of Paragonimus it has been found that the encysted cercariæ do migrate through the gills of the crab into water, and may be taken up by the definitive host from the water, but from what I have seen in other cases of encysted cercariæ in fish, a more probable explanation of the mode by which this Australian became infected is that he had some fish "sauce," or ate some uncooked fish during his visit to China.

THE HISTORY OF THE USE OF INTRAVENOUS INJECTIONS OF TARTAR EMETIC (*ANTIMONIUM TARTARATUM*) IN TROPICAL MEDICINE.

BY

GEORGE C. LOW, M.A., M.D., C.M.

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The history of any important discovery is always interesting, and that of giving antimony in large doses intravenously for different tropical diseases, is no exception to the rule. I hold no special claim to write this matter up, but having had occasion lately to consult the literature of the subject, before giving such injections in a case of ulcerating granuloma, I have thought that I might as well put the facts as I found them down, so as to save others the trouble and labour of wading through the references for themselves.

To NICOLLE and MENSIL[1] the honour is due for first proposing the use of antimony salts in tropical medicine, namely, in trypanosomiasis. Atoxyl and other arsenicals were then being largely used, both for experimental trypanosomiasis in animals and in human cases, and these authors not being entirely satisfied by the results obtained, suggested that antimony might be given a trial.

PLIMMER and THOMSON[2] followed this up and gave subcutaneous injections, of both the sodium and potassium tartarate of antimony, to experimental rats heavily infected with trypanosomes, and were at once struck with the wonderful sterilizing effect the drug had upon these parasites. Before treatment, a drop of blood examined microscopically, might be simply swarming with trypanosomes, but after one or two injections these had entirely disappeared.

Sir PATRICK MANSON[3] considered these results of such promise that he immediately gave two human cases, which he had under his care, similar subcutaneous injections, but unfortunately the local reaction of the drug, given in this way, was so severe that they could not be continued. He then tried giving it by the mouth.

About the same time BRODEN and RODHAIN[4] on the Congo tried giving the tartar emetic intravenously, natives suffering from sleeping sickness being treated in this manner. Naturally to begin with, as no one could know what exactly would happen, very small doses were employed, but as no abnormal effects followed, these were gradually increased until quite large amounts of the drug were taken. Clinically great improvement in the symptoms was noticed, and the difficulty of giving the antimony—a difficulty which had existed both by the oral and subcutaneous method of administration—was thus overcome.

LEBŒUF[5] claims that he independently discovered the intravenous route, and also gave injections by this channel about the same time as the authors just quoted. To these workers then the priority of giving antimony salts by intravenous injection belongs, and all subsequent work, both in trypanosomiasis, ulcerating granuloma and leishmaniasis, is based on their very valuable discovery. Some time after this MARTIN and DARRÉ[6] tried a combined treatment of atoxyl subcutaneously and antimony intravenously in a series of white men suffering from trypanosomiasis; very good results were again obtained.

Still later, KERANDEL,[7] who unfortunately had acquired trypanosomiasis himself, published his own case. At first treated on atoxyl he did not improve, so he next tried antimony by the mouth, having heard of the cases Sir PATRICK MANSON had treated in this manner. The drug caused sickness and could not be tolerated, so nothing remained but to try the intravenous administration. MARTIN and DARRÉ carried this out at the hospital of the Pasteur Institute with complete success, a cure resulting. Since that date a combined treatment by atoxyl and intravenous antimony (tartar emetic) has been carried out on most of the human trypanosome cases that have passed through the London School of Tropical Medicine,[8] and such treatment has been the standard one for this disease for many years.

The first records of the use of tartar emetic injections in leishmaniasis were made at the Brazilian Society of Dermatology in 1913, when

MACHADO and VIANNA[9] shewed cases treated in this manner. The case shewn by MACHADO was a woman aged 60 years. The WASSERMANN reaction in this case was positive, and though no leishmania were found, nevertheless the diagnosis of this disease was made, and the patient was given intravenous injections of tartar emetic. A complete cure resulted. The case exhibited by VIANNA also shewed good results from the intravenous use of the drug.

In the same year (1913) ARAGAO and VIANNA[25] treated cases of ulcerating granuloma by the same method with excellent results. In one case they gave twenty-one injections as follows:—First day, 0·05 of a gramme; second, 0·08 of a gramme; third to thirteenth, 0·1 of a gramme; fourteenth to seventeenth, 0·08 of a gramme; eighteenth to twenty-first, 0·1 of a gramme; or a total of about 1·8 grammes in all. In another case, a woman, ten injections were given within a period of thirty days. Rapid healing of the lesions took place in both these cases, the results being permanent.

TERRA and RABELLO[26] confirmed this work, using the same method of treatment for their cases. Intravenous injections of a solution of tartar emetic, dissolved in physiological saline, were given, one every day at first and then one on alternate days; after a few injections the ulcers began to shew signs of healing and completely disappeared after the fifteenth injection. According to their experiences tartar emetic is a specific for this disease.

During 1914, further papers on the treatment of cutaneous leishmaniasis by intravenous injections of tartar emetic were published by DA SILVA[10] and CARINI,[11] in Brazil.

Turning now to the New World, it was apparent to all that such injections might be useful for leishmaniasis as seen in the Mediterranean Basin and India; and in the latter part of 1914, CASTELLANI[13] treated a case of kala azar in this way in Ceylon, while DI CRISTINA and CARONIA[12] similarly used the method for the form of leishmaniasis found in the Mediterranean area (infantile kala azar). About this time, or a little later, ROGERS,[14] MACKIE,[18] and MUIR,[16] began to systematically treat Indian cases of kala azar by tartar emetic injections, obtaining results much more successful than by previous treatments. ROGERS in his paper apparently claims to have independently discovered this line of treatment for kala azar, but if the facts just detailed are closely studied

such a claim cannot stand. WENYON, in a summary of ROGERS's paper (Trop. Dis. Bul., Vol. VI., p. 221), rightly states the position as follows:—

> "A Notice of GASPAR VIANNA'S Treatment of American Cutaneous Leishmaniasis by means of Tartar Emetic appeared in this Bulletin February 14th, 1914. It is to this observer that the credit of first employing the drug intravenously in leishmaniasis is due, and to DI CRISTINA and CARONIA for using it in kala azar with success, must be given the credit of priority, even though the possibility of employing the drug in kala azar had previously been in the minds of most people having any knowledge of the disease."

This is correct as regards the Mediterranean form of the disease, and if any priority is necessary for the application of a well-known method of treatment to another disease, this would, for the Indian form of kala azar, as far as publication is concerned, go to CASTELLANI.

The results obtained by antimony in the three diseases quoted in this paper are in many ways good, and far in advance of other drugs that have already been employed; but at the same time it is necessary to add a word of warning. Its use has not prevented a high mortality amongst the Rhodesian and Nyasaland cases of human trypanosomiasis, and it remains to be seen whether it will act as a complete specific for all cases of leishmaniasis. As regards ulcerating granuloma, many cases are cured, but there still seems to be a small minority, which, though benefitted by the administration, are not absolutely cured. A case recently published by Dr. NEWHAM and myself[29] did very well, a cure resulting; but now we have a similar case in which though large numbers of injections of antimony have been given, no definite cure has been brought about. Still, a certain degree of improvement has taken place, and by a combined therapy of antimony and X-rays, we hope the desired result will be obtained.

The possibility of the use of antimony intravenously in other diseases, both in the tropics and in temperate regions, should not be lost sight of, in view of the results so far obtained in the diseases in which it has been employed. It might, for example, be given a trial in malaria and in other affections, especially of a protozoal nature.

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This list of references does not of course claim to be a complete one. It aims, as far as possible, at giving the chief or salient points of the history.

NOTES AND COMMENTS.

QUININE AS A PROPHYLACTIC.

BY

CAPT. C. F. HARFORD, R.A.M.C.

To my great regret I was unable to be present at the meeting of the Society on October 20th, to hear Dr. MACDONALD'S paper. If I had been there I should certainly have demurred to his statement of the case with reference to quinine, as it appears to me to be certainly misleading.

Quinine prophylaxis has been debated on many occasions in the history of the Society, and there has been overwhelming evidence in its favour. To recite this over again would mean elaborate references to tropical literature, and I am away from all such literature.

The pity is that those who criticise do not appear to recognise the position taken up by the advocates of quinine prophylaxis. Let me, then, state the position which I believe would be generally conceded.

1. There is no suggestion in these days that quinine prophylaxis should take the place of anti-mosquito measures. On the contrary, many who believe strongly in the importance of the former have been among the strongest supporters of anti-mosquito work.

Students trained by me in the elements of tropical hygiene have done splendid work in the elimination of mosquito breeding-places in Central Africa and elsewhere, yet they are strongly in favour of the daily dose of quinine where necessary. I have also written frequently on anti-mosquito work.

Sir WILLIAM MACGREGOR was one of the chief pioneers in the education of the Nigerian natives as to the rôle of the mosquito in the spread of malaria, which Dr. MACDONALD very properly advocates, yet he believed most strongly in the proper use of quinine. It was largely due to his influence that Europeans at work in West Africa adopted, to a very great extent, quinine prophylaxis, with the best results.

2. I am not aware that anyone contends that quinine prevents infection. What is contended is that quinine is the only agent known

which is antagonistic to the development of malarial parasites existing in the blood, so that where an individual has been infected an attack of fever may be prevented, or if one attack has occurred subsequent attacks may be warded off.

I do not think that anyone would be surprised at the failure of quinine to ward off fever under the conditions referred to by Sir DAVID BRUCE.

3. As to the suggested evil effects of quinine taken in the usual prophylactic doses, I have enquired very extensively into these, and, except in the case of idiosyncrasy, which is found in the use of most drugs, I do not believe that quinine is in any way injurious.

I am exceedingly sorry that the subject of blackwater fever has been brought up again without a particle of new evidence.

I consider that the great reduction in the incidence of blackwater fever in West Africa is largely due to quinine prophylaxis; but here, again, the marshalling of the facts which could be adduced in support of this view would occupy much space, and need reference to material which is not available.

So much for the main points at issue with reference to quinine prophylaxis.

Dr. MACDONALD says: "Quinine relieves suffering, and by destroying parasites limits the number of infective gametocytes." This is valuable testimony; but it is a pity that the previous sentence seems to discourage the use of quinine even in treatment, for he writes: "Large masses of peoples without taking quinine recover from malaria between each seasonal infection." Of course they do, as do people from most diseases without use of the appropriate remedy, but what of the mortality and suffering involved?

Dr. MACDONALD will, I hope, forgive these criticisms. We are all ready to support him in advocating the supply of an adequate sanitary staff in every colony, and probably we should agree that in the ordinary sense quinine prophylaxis, as regards natives at least, should not be dealt with by the sanitary administration. For Europeans, however, particularly in Central Africa, East and West, with its abundance of malignant parasites, there is good cause for advocating the use of a daily dose of quinine of five grains, which has in the past produced most important results.

Obituary.

We regret to announce the death on September the 8th, 1916, of Dr. R. H. Von Ezdorf, Surgeon U.S.P.H. Service, U.S. Marine Hospital, Mobile, Alabama, a Fellow of the Society of Tropical Medicine and Hygiene. Dr. Von Ezdorf did much useful work in Tropical Medicine, especially as regards malaria in the United States. These results were usually published in the U.S. Public Health Reports, his most recent papers being "Anopheline Surveys"; "Methods of Conduct and Relation to Anti-malarial Work"; "Malaria in the United States: Its Prevalence and Geographical Distribution"; and "Demonstrations of Malaria Control," the latter appearing in March, 1916. Dr. Von Ezdorf died suddenly, and was working right up to the end. His death is a loss to Tropical Medicine, and will be universally regretted by all workers in this branch of the profession.

G. C. L.

TRANSACTIONS

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THE VOMITING SICKNESS OF JAMAICA.

BY

H. HAROLD SCOTT, M.D., M.R.C.P. (LOND.), D.P.H.

Government Bacteriologist, etc.

My endeavour this afternoon will be to place before you a few facts concerning one of the most interesting diseases occurring in warm climates. At present it affects only a small part of our tropical possessions. No record of it is to be found in the majority of books on tropical diseases; in one well-known work there are a few lines, mostly wrong. As regards any definite findings as to the nature and causation of the condition, the affection spoken of hitherto as the Vomiting Sickness of Jamaica must be looked upon as a new disease. I will attempt in the time allotted to me to put you in possession of certain facts, which it has been my good fortune to work out and, in some cases, discover.

First, a few general remarks on the so-called Vomiting Sickness of Jamaica. The disease has existed in that island for many years, particularly prevalent in the cooler months, November to March or April,

and in each year it has been responsible for a considerable number of deaths, and in some years has exacted a terrible toll among children.

The earliest records which I have been able to trace date from thirty years ago, when the non-committal but descriptive name of "the Vomiting Sickness" was given to a condition which was beginning to command respect owing to its mysterious nature, its sudden onset, and its high mortality rate (80 to 90 per cent.).

Tracing the history of the disease from that time (1886) to the present has been an interesting study, but it must be passed over to enable us to come to more important matters. Briefly, the period 1886 to 1915 may be summed up by saying that, during the season in which the disease was usually prevalent, any child that died after vomiting was diagnosed (by some practitioners) as having suffered from vomiting sickness, while other medical men, rightly scorning so indefinite a term, erred in signing up true vomiting sickness cases as having died from gastritis, enteritis, worms, malaria, cerebro-spinal meningitis, yellow fever, and so on; while yet a third group—fortunately a small one—on attending patients with some obscure condition terminating fatally, would sign the certificate by the delightfully safe but paradoxical diagnosis, "vomiting sickness without vomiting," or, if of a classical turn of mind and desirous of impressing the relatives, "vomiting sickness *sine vomitu*." The name was, in fact, as serviceable as charity in the multitude of sins which it sufficed to cover.

In 1906, letters were sent to the other West Indian Islands, asking whether any such or similar disease was met with there, and the replies all went to prove that the condition is practically limited to Jamaica.

So serious were the ravages of the disease that, in 1910, an expedition was sent out from England to investigate it, but without result, and again, in 1912, a second expedition was made, which, in turn, came back baffled.

This was the state of things then, a veritable chaos, when, in February, 1915, a severe outbreak occurred on the north side of the island, and eighteen deaths took place in a small district in two days, and I was sent down to investigate the matter on the spot.

I had the good fortune to see a considerable number of cases, some almost from start to finish, and to perform autopsies on all who died during my stay in the district. I visited the huts where cases had

occurred, and, by the kindness of the District Medical Officer, was taken to interview survivors and the relatives of those who had died. There will not be time to go into the details of the many cases seen there; a brief summary will be given later, but before doing so it will be advantageous to describe to you shortly the symptoms of the condition. The usual sequence of events is as follows:—

The patient—almost invariably a child—in apparently perfect health, suddenly complains of feeling ill, and occasionally of pain in the stomach. This is usually mere discomfort and not, I believe, actual pain, but, as the native tersely puts it, "him belly trouble him, doctor." He then vomits; perhaps only once, perhaps three or four times at short intervals. Recovery then seems to take place, and, if the attack occurs at night, the child drops off to sleep, apparently well again. Some three or four hours later (occasionally after a longer interval) the child wakes up, again complains of feeling sick, and begins once more to vomit, usually frothy mucus, and later watery fluid only, or it may be bile-stained. There is little if any accompanying effort, unless the stomach be empty, when troublesome retching may ensue. Within a very short time, often a matter of a few minutes only, convulsions make their appearance, coma rapidly supervenes, and terminates in death.

Recovery from the first attack of vomiting being to all appearances complete, a doctor is not usually summoned until the relapse occurs. The majority of patients, therefore, first come under observation during the convulsive or comatose stage. The temperature is usually normal or subnormal, rarely 100° or 101° F.; the pulse is of good volume, and rate is 90 to 100 per minute; respiration 26 to 30, and regular until towards the end, when the Cheyne-Stokes rhythm may be noticed. The pupils are equal, moderately dilated, and, if the coma is not too deep, react to light. There is no delirium, and, shortly before passing into the comatose stage, the child may remark that it feels very bad, but does not call attention to any particular symptom or complain of any localised pain. There is no rigidity in the true vomiting sickness cases (except, of course, during a convulsion) but a general limpness of muscles; movement, such as turning the patient over for examination purposes, or to obtain fluid by lumbar puncture, frequently leads to a repetition of the vomiting.

Such is the picture of the majority (80 to 90 per cent.) of such cases, for the mortality rate is very high. In the rare instances which recover,

the vomiting is practically the only symptom. I have never met with a recovery when once convulsions or coma has set in. Recovery when it occurs is very rapid. Within twenty-four hours or so, a child who had appeared to be seriously ill may be up and about, shewing nothing but a little pallor and debility, as after any severe bout of vomiting; while others in the family, who did not seem any worse at the time, have passed into a state of coma and died in a few hours.

I crave your attention while we deal with a view of the symptoms in a little more detail.

1. *Vomiting.*—This may be, and usually is, the first objective symptom. The nature of the material vomited is usually at first food or if a considerable interval has elapsed since the last meal (and this is uncommon), frothy mucus, then watery matter, later bile-stained. At times the vomiting may be replaced by troublesome retching. Next, as regards the times at which vomiting occurs. In a typical case, as you have already heard, vomiting takes place at the onset, and may be the very first symptom. It is usually accompanied by considerable effort and is repeated two or three times, at short intervals. This I have termed the "initial vomiting," and gives one distinctly the idea of an effort on the part of the stomach to rid itself of some noxious material.

After an interval of calm, during which there are practically no symptoms, there is a return of the vomiting, and now it is of a different character. It is, to a great extent, effortless, and may be unaccompanied, by nausea. This I have designated the "secondary vomiting," and it is in my opinion, cerebral in origin, owing to its character of being effortless and induced by movement, and to its being followed almost at once by other nervous symptoms, twitchings, convulsions, and coma.

Initial or secondary vomiting may be absent. Thus, as stated already, the former only is seen in cases which recover—in other words, the patients never reach the stage when the secondary cerebral symptoms appear. On the other hand, this "initial vomiting" may be suppressed in the very rapid and acute cases. There is an attack of vomiting which is so rapidly followed as to be almost accompanied by the convulsions and coma, the entire symptoms being cerebral, owing, as I interpret it, to rapid absorption of the toxin from an empty stomach. Death in these cases may occur in an hour, or even less.

More rarely, the secondary attack of vomiting is suppressed; the

patient may pass through the initial attack and appear to improve; then, after a considerable but varying interval, he is seized with convulsions, passes into the comatose stage and dies.

Lastly, vomiting may be absent, and the cerebral symptoms may be the first indications of anything wrong. For example, I have among my notes the following case:—A child, four years of age, was quite well when she ate her dinner at 1 p.m. Two hours later (note the interval, please), she felt out of sorts and went to lie down. An hour or so afterwards her mother went to waken her, but could only partially do so; twitching of limbs and slight convulsions came on, and the child lost consciousness altogether and remained comatose till death.

Such cases have been diagnosed somewhat paradoxically as "vomiting sickness without vomiting," and I am sure that they do occur, though with exceeding rarity. Such a diagnosis, however, could not be made unless, firstly, true vomiting sickness cases were occurring at the time; secondly, all other causes could be excluded; or thirdly, the post-mortem signs, especially the microscopical, were those of vomiting sickness.

2. *Convulsions.*—These may vary from merely slight twitching movements of the limbs to definite massive convulsions. They may be tonic muscular contractions lasting for a few seconds only, or more clonic and epileptoid in character. Looking over my notes in cases which recovered, slight twitching movements occurred in one patient only, a child of four years of age. We have all of us seen slight twitching movements in a child asleep, in ordinary health, or possibly with a little dyspeptic disorder. With reference to the vomiting sickness one may safely say that in no cases which recover are convulsions seen.

3. *Coma.*—In most of the cases which I have seen this has been deep; as a rule, there was absolute unconsciousness with absent conjunctival reflex. In some, at an earlier stage of the coma, there was general flexion, and some irritability was exhibited when attempts were made to rouse the patient; but this "cerebral irritation stage" was transitory and soon passed into one of deep coma.

A few remarks may be added on the questions of age, sex, and duration of illness.

Age.—The condition is, to a great extent, one of childhood; sucklings are not attacked. I have records of only two cases under the age of twelve months, and these were not breast-fed. Nearly half the cases

(44·84 per cent. of my series) occur in the first quinquennium, another 30 per cent. in the second, and 85 per cent. of cases are under the age of fifteen years. The mortality rate is high in all these periods; thus, of those under five years, 85·06 per cent. died; of those between five and ten years, 86·21 per cent. died; and 84·24 per cent. of those attacked under fifteen years succumbed.

Sex.—The affection shews no predilection for sex. Thus, in the first decade, 45 per cent. of those attacked were males and 55 per cent. females; and the death-rate was closely similar, 46 per cent. and 54 per cent.; while in the third quinquennial period, of 58 cases of which I have full notes, 30 were males and 28 females; of these, 50 terminated fatally, and this number was contributed to equally (25 each) by males and females.

Duration of illness.—In 140 instances I have been able to obtain reliable accounts of the duration of illness. The shortest recorded was in a female child of one year, death taking place in thirty-five minutes. The average duration of the total number works out at 12·72 hours. Sex has no influence on duration, for, although of those whose duration is given 82 were females and 58 males, the length of illness from the time of onset to death—including, when present, the period of calm— works out at 12·5 hours in the case of males and at 12·89 hours in females, a difference of only 23 minutes.

It will not be time wasted if I briefly recapitulate the symptoms by shortly describing four cases exhibiting the different types:—

1. A mild case: a girl, nine years of age, was given some "soup" from boiled ackees and bananas at noon. At 2 p.m. she complained of pain in the belly and vomited. This vomiting continued on and off for three hours. She was seen by a medical man, who gave her a mixture containing ether and ammonia. The vomiting ceased, and the child had quite recovered by the following evening.

2. A male, aged three years, in normal health when he was given an evening meal of the soup made from bananas, yams and ackees. Two hours later, without any complaint of pain, he vomited. He rapidly recovered from this, and appeared quite well on being put to bed an hour or so later, and slept well till just before dawn. He then, without any warning, suddenly vomited again, was shortly afterwards seized with convulsions, and coma supervened, which lasted till death at 11 a.m. The

total duration was 16 hours; there was a symptomless intermission of 8 to 10 hours, and death occurred in 5 hours after the onset of the secondary vomiting. Here we have an example of a case apparently quite mild at first, but nevertheless terminating fatally.

3. A girl of six years, after a similar meal, went to bed in her usual good health. Early in the morning, without any warning or previous complaint, she suddenly vomited, and did so three times in the course of an hour. During the day she stayed in the house and did not feel quite well, but took food. She seemed better in the evening, and slept well during the night. Early the following morning, without warning, again she started vomiting, frothy watery material, without any effort. A few minutes later she was attacked by convulsions and passed into a state of coma, dying at 2 p.m. This case resembles the last, but differs in the longer duration of illness, and in the fact that the interval was one of improvement, not total abatement of symptoms.

4. A girl of twelve years left home in good health for school, three miles away. At midday she had a meal containing ackees, and returned to school, where nothing amiss was noticed, until three hours later she started to vomit; this occurred four times. Feeling better she started for home, but during the walk she felt ill again and vomited at intervals, taking three hours to make the three-mile journey. Shortly after arriving she became drowsy, this drowsiness deepened to coma, and she died about midnight without recovering consciousness. Here we have an example of a case in which convulsions were absent.

The rare condition of "vomiting sickness without vomiting" has already been described.

Let us now pass on to the pathological findings. I will not weary you with a long description of these. To describe them in detail would occupy more than the whole of the time at my disposal this afternoon. I have brought a series of specimens, some of which you will find under the microscopes, and I shall be very pleased to point out any of the special features to those who desire to know more of this interesting subject. The changes set up are very widespread, and may be briefly summarised as follows:—

Hyperæmia of most of the organs, including the meninges, with œdema of the supporting tissues; there is a tendency to hæmorrhages evidenced by small extravasations, *e.g.*, in kidneys, adrenals, lymph

glands, spleen, lung ; the epithelium, particularly of the kidney tubules, the pancreas and liver, shews necrobiotic changes ; and, lastly, and most important, is a marked fatty change in many organs, notably the liver, kidneys, sometimes in the cells of the pancreas and heart muscle, and the large Betz-cells and others in the brain.

Full description of the morbid anatomy, macroscopical and microscopical, is given in my larger paper on this affection published in the *Annals of Tropical Medicine and Parasitology* earlier this year.

Of course, in a disease such as this, which becomes epidemic every year, bacteria have been suspected and carefully looked for, without result. In fact, two years ago, during the course of my investigations, I stated " in my opinion the disease has no bacteriology. The organisms which have been found in some of the patients (a small percentage only) I can see no reason for regarding as in any way causative." The absence of prodromata, of any true incubation period, the serious and extensive lesions, the negative results of attempts frequently repeated at finding or isolating any organisms, all made me incline to the opinion that the condition was not a bacterial infection, but a pure intoxication, and we shall see how the sequel bore out the surmise.

We will now return to the outbreak on the north side of the island, of which mention was made earlier in the afternoon, for this was the turning point of the whole investigation, and after that events marched rapidly.

I investigated 32 cases during my stay in the district. A few of them may be briefly mentioned in order to make clear to you the sequence of events and the direction which my investigations subsequently took.

1. A male child, aged ten years. At 5 p.m., February 17th, complained of pain in the stomach and began to vomit. He vomited at intervals till 1 a.m., 18th, when convulsions set in, rapidly succeeded by coma, which lasted till death at noon. This patient had been quite well till the first vomiting at 5 p.m. At 3 o'clock, two hours previously, he had partaken of a meal consisting of yam, ackees, and bananas, all boiled together. The ackees were mostly removed and eaten by the older members of the family, leaving the soup, or " pot-water " as it is called, for this child and the one following, who, in addition to taking some of the soup, was given a little of the solid also.

2. Female, thirteen years ; cousin to the last, and living in the same

house. Also quite well until about 5 p.m., when she began to vomit and continued to do so off and on till midnight. In the course of the following morning vomiting again returned, and the patient soon after lapsed into coma, and death took place during the succeeding night. While comatose this child was restless, but did not have any definite convulsions.

3. The mother of the former and aunt of the latter, aged twenty-six years. Shared in the same meal, but only ate the solid ingredients (and naturally some absorbed fluid). Vomiting started about the same time (5 p.m.) and continued during the night, but ceased in the early hours of the morning. Later on, feeling better, she took some more of the same articles of food. Vomiting set in again the same evening and persisted at intervals during the following day. There was no convulsion, no coma, and the patient recovered in 24 to 36 hours, though she felt weak.

Two other adults, aged twenty-nine and sixty-five respectively, of the same family, suffered similarly to the last, but recovered in the same way.

I would ask you to notice in this series that the first meal was shared in by all; the subsequent one contained the same ingredients. The adults ate the solid and recovered after pretty violent attacks of vomiting; the older child had some of the solid and some of the soup and died after 34 hours; the younger had soup only and died in half that time; and the interval between the meal and the onset of the vomiting was in each case about two hours.

One more series, this time of three. A girl of eight years, a boy of two years, and their mother, twenty-five years of age. All partook of a meal consisting of yam, banana, pumpkin and ackee boiled together. The other child had a little of the solid with some of the "pot-water." She suffered with vomiting, succeeded by convulsions and coma, and died in 16 hours. The younger had the soup only, suffered with the same symptoms but more acutely, and died in two hours. The mother ate the solid ingredients, was acutely ill with vomiting, so ill in fact that, as she expressed to me with tears in her eyes, she was unable to attend to her children who were dying. Her attack of vomiting continued for nearly 24 hours, after which she rapidly recovered.

If I may, without trespassing too greatly on your indulgence, I would like to quote one last case:—

A child, a little girl of three years of age, at 11 a.m., February 24th,

was given the liquid or "pot-water" made from yam, peas and ackee. At 1 p.m., two hours later, please notice, without any symptom or complaint during the interval, she vomited twice and went to lie down. At 2 p.m. the attack recurred, muscular twitchings and convulsive movements supervened, consciousness was lost and the child died comatose at 5 p.m., the total duration of illness being only four hours.

I will not weary you by further narration of cases. I have notes of between 300 and 400 now, and could quote several such series as those just detailed.

To sum up the 32 cases which came under my notice in this outbreak: in 17 the attack followed closely on the ingestion of ackees or a watery extract (soup or pot-water) made from them. In eight others there was a strong probability that ackees comprised one of the constituents of the meal prior to the onset of the illness. In these instances the parents had had a meal containing ackees and the children "may have had some." In the remaining seven cases no definite history of the food could be obtained; but I visited the huts in which the cases had occurred and noticed in every instance, without exception, that trees bearing ripe fruit were growing in the yards, and it is most unlikely, to say the least, that such a food, ready at their very doors, a food of which they all are fond, and which was then ripe would be avoided, especially at a time when other articles of food are scarce or at least relatively expensive. In none of the 32 then could the eating of the fruit be definitely excluded.

The next point was to make enquiries concerning the ackee, the fruit of *Blighia sapida*, which is used to a considerable extent as an article of diet in Jamaica. Amongst the better classes the ackees are gathered carefully, one by one, and only those which are properly opened and appear ripe and sound in every way are taken for food. Unopened ackees are not used by such people, nor any which have not opened naturally on the tree or have been gathered from an uninjured branch; those forced open after falling from the tree unopened are dangerous. Among the poorer people, however, less care is taken, and a boy is sent up the tree to shake down the fruit; ripe and opened and unripe unopened fall together; the former is collected and the latter left. In time, some of these may open and be gathered with fresh ripe ackees brought down at the next shaking.

By the time the investigations into the Montego Bay outbreak and the examination of the various tissues taken post-mortem were nearing completion, I considered that sufficient evidence had been presented to warrant the bringing in of a true bill against the ackee, sufficient, that is, to put it on its trial, so experimental work was started with this end in view.

Details of my earlier experiments and their disappointing barrenness of results have been given in the monograph already alluded to The reasons for their failure were disclosed subsequently, but it would be profitless now to relate either the experiments or the causes of their failure. I will merely deal briefly with those which arose directly out of the indications obtained from a study of the outbreak described above. The aim in view as you will have gathered, was to establish whether any, and if so what, connection existed between vomiting sickness and ackee poisoning.

In order to simulate as closely as possible the conditions under which, by this hypothesis, cases of vomiting sickness occur, some ackees were obtained which to all appearances were good except that they were unopened or had been forced open after being gathered. The part used for food was then boiled with water just as was done by the natives in making their soup or "pot-water." The product, practically a watery extract of ackee, was then filtered. The result is a liquid of the colour of weak tea with a layer of oily, fatty matter like melted butter floating on the surface.

This extract was administered by mouth to kittens, a pup, guinea-pigs, rabbits, Belgian hares. The three last-named were unaffected. The other animals exhibited the same train of symptoms, so that the recital of one will suffice for all. Within an hour of administration of a small quantity vomiting set in, and the animal was inclined to be heavy and dull for about half to one hour. Recovery then took place and the animal became normal and lively again. The following day a slightly larger dose was given with similar results, recovery being apparently complete in two hours or a little more. After an interval of three hours a third dose was given twice the size of the first. Vomiting came on 45 minutes later, and the animal became dull and drowsy, its head nodding as with sleep in some cases, in others it merely lay about and was disinclined to move and vomited at intervals. This drowsiness

gradually deepened to coma during the succeeding hour, and death took place some four hours after the last administration. The total amount given was the extract from one ackee. The post-mortem appearances were, both naked eye and microscopically, absolutely typical of those found in human vomiting sickness patients. I have the specimens here for you to see this afternoon. I repeated the experiments on several animals, merely varying the dosage, and except for a slight difference of interval between the feeding and the onset of the vomiting, according as the dose was small or a little larger, the symptoms and post-mortem appearances were the same.

Briefly stated, the characteristic symptoms of the so-called vomiting sickness appear an hour (more or less) after the administration of filtered watery extract of ackee. In human cases, where other food was taken as well and the action probably slower in consequence, the interval was usually two hours. After a small dose there was vomiting, and after a larger still, vomiting, drowsiness, coma and death.

The matter had by this time progressed beyond the realm of mere hypothesis, and a most welcome confirmation came three months later, when the following case occurred:—

On the evening of August 19th, 1915, a family of eight, all at the time in good health, partook of a meal of ackees taken from a branch of a tree which had been damaged by the hurricane of the previous week. About two hours later, five of them complained of feeling sick; later three of these were attacked by vomiting, and one who had drunk some of the soup, shortly afterwards became convulsed, rapidly lost consciousness, and died within 24 hours of the meal. The remainder completely recovered.

Six days afterwards, at 6 p.m., another similar meal was prepared. The soup together with some of the boiled ackees were eaten by a woman twenty-four years of age. At 8 p.m., she vomited and soon afterwards stated that she felt better; at 10 p.m., however, the vomiting returned, convulsions followed, coma set in and death took place shortly after midnight. Another member of the family was also taken ill, but recovered after vomiting. The autopsy I carried out myself and took specimens of practically every organ and tissue. Full details of both macroscopical and microscopical appearances have been given in the paper already spoken of, and I have brought sections with me to-day.

Here then was a definite history of a patient previously in good health partaking of a meal of ackees from a bruised limb. She, with other members of the family, suffered from vomiting and recovered. A week later another meal was prepared with fruit from the same tree. The patient drank the soup and also ate some of the solid. Two hours later the symptoms appeared and ran their course to fatal termination in six hours or so, and at the post-mortem the changes were revealed which have been mentioned earlier, and which you can see for yourselves afterwards. In this case the term "vomiting sickness" was not used from first to last, but the case shewed typically the onset, course, and pathological changes of that disease.

Certain peculiarities and characteristics of the affection which at the outset were most puzzling, find a ready explanation in the light of our present knowledge of the similarity (may one say identity?) between vomiting sickness on the one hand, and the effects and results of experimental administration of ackee extract on the other, linked together by the clinical case of definite ackee poisoning just related. These were:—

1. *The peculiar seasonal prevalence.*—The epidemic character of the disease corresponds exactly with the main ackee season, when other fruits and natural foods are relatively scarce. If the ackee season lasts longer than the usual November-December to March-April, then also cases of vomiting sickness continue to be reported for similarly longer periods. Ackees are also obtainable in smaller quantities at other times, but other foods are then plentiful and this fruit is less eaten. Occasional cases of vomiting sickness, however, appear at other times as the one just related. It used to be thought that it was a disease of which occasional, sporadic cases occurred during the warmer months, becoming epidemic in the cooler, comparable, for example, with cerebro-spinal fever due to the meningococcus.

2. *Limitation to Jamaica.*—The results of the circular-letter sent to the authorities of other West Indian islands have already been mentioned. I myself have made enquiries of inhabitants of other islands and am told that the *Blighia sapida* does not grow to any extent in any of them. It is true that one or two trees are found, for example, in St. Lucia and, I think, Barbados, but they are looked upon as curiosities and are not used for food.

3. *Sudden onset of symptoms* in the midst of apparent good health,

without any incubation period or prodromata, and in the well nourished and not necessarily the emaciated or debilitated. We see now that the symptoms, being those of an acute intoxication, would depend not so much on the general well-being of the subject as on the dose of the poison and the condition of the stomach, whether empty or full, and its consequent readiness for absorption.

4. *The rapid and complete recovery of non-fatal cases.*—This is obvious, and explained by the fact that an acute vegetable poison is taken; if the dose is small it is got rid of by the vomiting, and the patient recovers.

5. *Affection of several persons practically simultaneously in one house* or close neighbours in a settlement. Several members are affected in one house because the food is cooked together and shared in common. Close neighbours in a settlement are affected because the trees are in and about the settlement and all share in the produce.

6. *The vastly greater preponderance in children.*—This is explained by the fact that they are given the "pot-water," the most toxic part—an extracted poison, in short—and that the lethal dose of a poison is far smaller for a child than for an adult; and also the adults know the risks of eating unopened ackees while children naturally do not.

7. *Attacking the West Indian native in much greater numbers than the East Indian or the white man.*—In Jamaica the coolies live largely on rice and split peas, often in the form of curry; they also like green fruit—mangoes, guavas, jack-fruit. They rarely indeed eat ackees. A few, after they have served their time and settle in Jamaica, may eat them, but not at all commonly. The white buys his ackees in the market, where he can see and select them; while, safer still, many will only eat ackees which have been carefully gathered under their own superintendence and from their own trees.

Further investigation I hope to undertake later on to elucidate, firstly, the actual constitution and nature of the poisonous ingredient. Whatever its nature it appears to be rendered inert, partly if not completely, by alcohol. Patients seen in quite an early stage, the initial (gastric) vomiting period, had the best chance of recovering on the administration of stimulant—ether, rum, whisky, brandy. This is further borne out by the fact that extracts made with alcohol, ether, or petroleum

ether were, as far as I have been able to test them, innocuous, while the watery extract, as you have seen, proved rapidly fatal.

Secondly, to try to find an antidote, though the action is so rapid that one cannot hope much from it, even if discovered; and, thirdly, to try to explain why this substance should act so energetically on carnivora—human subjects, kittens, dogs—and have no effect on herbivora intragastrically. I have administered to guinea-pigs and rabbits several times the quantity of the same extract as sufficed to kill a kitten, but without any obvious untoward effect.

I may add that on the completion of the investigations which have been related, I was asked to draw up a notice of precaution as to the use of this fruit as a food. This was done shortly before my departure from Jamaica last year, and I have had a letter from the Governor, and also from others in the island, telling me that the cases of vomiting sickness last season were exceptionally few.

To sum up:

1. The term "vomiting sickness" has been used in Jamaica for many years as a comprehensive name for various diseases, including cerebro-spinal meningitis, gastritis, gastro-enteritis, worms, malaria; in fact, any disease occurring in the cooler months and associated with vomiting and convulsions.

2. During the last ten years the idea has been gaining ground that there is an affection included under the term "vomiting sickness," whose course of symptoms and post-mortem changes are not those of any known disease.

3. The death-rate from this affection is exceedingly high, 80 to 90 per cent., and a fatal termination takes place in a few hours.

4. Investigations into a typical and severe outbreak in February, 1915, revealed the fact that, in a majority of the cases in which a reliable history was obtainable, ackees formed part of the last meal taken in health, and that this article of food could not be excluded in a single case.

5. Persons drinking the soup or "pot-water" made with ackees in certain conditions shewed the most acute symptoms; the onset occurred in about two hours, and death nearly always resulted.

6. The fruit is poisonous if picked from a decayed, bruised or broken branch; if forced open and not opened naturally on the tree, amongst other conditions.

7. Much of the poison is extracted by boiling with water. .

8. The symptoms of a case of typical vomiting sickness are: initial vomiting (gastric in origin) coming on in apparently perfect health; a period of improvement lasting a few hours, succeeded by secondary vomiting (cerebral), rapidly followed by convulsions, coma and death. The average total duration of illness is twelve and a half hours. Initial or secondary vomiting or convulsions may be absent, but not in a large percentage.

9. Recovery, in my experience, has never occurred when once convulsions have set in, or coma if convulsions are absent; and as a corollary to this, in no cases which recover are convulsions seen.

10. The affection is largely one of childhood, and shews no predilection for sex.

11. A reasonable interpretation of the symptoms is: some poison is taken, or some substance which acts as a poison after it enters the stomach. If the initial vomiting is able to get rid of this substance no further symptoms occur, and recovery is rapid. If this is not the case, there is an interval, a more or less quiescent period of absorption, after which there follow symptoms due to the action of the poison on the higher centres—secondary (cerebral) vomiting, convulsions, drowsiness, coma and death.

12. In rare instances the cerebral symptoms are those first noticed—convulsions, drowsiness, coma; there is no preceding vomiting—the so-called "vomiting sickness without vomiting."

13. Intragastric administration of an extract made by boiling unopened ackees with water produced in certain laboratory animals (kitten, dog) the symptoms and pathological changes seen in cases of vomiting sickness.

14. A case of ackee poisoning in a human subject exhibited the same symptoms, course and post-mortem changes, macroscopical and microscopical, as *(a)* human vomiting sickness cases, and *(b)* animals to whom an aqueous extract of unopened ackees had been administered.

15. The characteristics of vomiting sickness, the seasonal prevalence, the sudden onset in health, the rapid and complete recovery of non-fatal cases, the rarity of occurrence in white children and East Indians, the pathological changes set up, and so on, all find explanation in the view that the condition is an acute intoxication by the unwholesome ackees—the fruit of *Blighia sapida*.

Dr. G. C. Low: We must thank Dr. SCOTT very much indeed for coming and giving us this very interesting paper to-night. I am very sorry we have had such a poor audience to receive and listen to it, but, even though this is so, it will go out in the TRANSACTIONS, and I have no doubt that many members who have been unavoidably prevented from being here this evening will read it later with great interest and pleasure.

There are several points which, I think, Dr. SCOTT might follow out, or get followed out, further. For instance, why not have the fruit analysed, in order to ascertain what the poison in it is? It certainly seems very strange why the fruit from a bruised branch, or fruit which has been opened prematurely or unnaturally, should be poisonous, while the ripe fruit which has burst open of itself is not poisonous. It seems difficult to say exactly why this should be so, though, of course, it might be that the poison is a volatile substance—a substance which has escaped in the open ripe fruit, but not in the case of the immature fruit. I do not feel any doubt in my own mind, after hearing Dr. SCOTT's paper, that the poison of this fruit is the cause of all the symptoms from which these patients suffered.

Another interesting point, which might also be gone into in detail, is whether this fruit grows in the other West Indian Islands as well as in Jamaica. I would ask whether Dr. COCKIN has seen ackees in Grenada. If the fruit grows in other islands in the West Indies, do the natives there eat it? Also do they eat it in other parts of the tropics, such as the West Coast of Africa?

The microscopic specimens we have seen to-day are certainly very interesting and instructive; they shew very toxic changes, the fatty degeneration and lesions in the liver and other viscera being extreme.

It is quite conceivable that these might be mistaken for yellow fever, and it is clear that ackee poisoning must be considered in the differential diagnosis of that disease.

With regard to guinea-pigs, rabbits and other animals being immune from symptoms when the immature fruit is given them, whereas it has strong effects upon kittens and dogs, the same sort of thing is seen with other plants even at home here in England. Rabbits and hares may eat different shrubs with immunity, whereas if these were given to carnivores poisonous results would quickly follow.

Dr. R. P. COCKIN: With reference to Dr. Low's remarks concerning Grenada, I am in a position to state that ackees do exist there. They are, however, regarded—as, I believe, is also the case in St. Lucia and Barbados—as botanical curiosities, and the fruit is not eaten.

I would like to ask Dr. SCOTT if, during his experimental work on animals, the solid part of the ackee fruit was administered with the oily extract, with the view of ascertaining whether the solid part is capable of fixing the "oil," and so having an antidotal action.

I would also ask him if he can explain the marked degree of fatty degeneration which his stained sections shew takes place in the liver and other organs. The extent of this fatty degeneration is out of all proportion to the short duration of the illness, and I should be grateful for information on this point.

The CHAIRMAN (Sir DAVID BRUCE): We must all heartily congratulate Dr. SCOTT on having brought this investigation to a successful termination. Many other men attempted to solve this problem, but all failed. It may be said that the solution is a simple one, and might have been solved by anyone who could look beyond his nose. But this simplicity is the characteristic of all great inventions and discoveries, and it would seem the power to look beyond their own noses is given to few. Dr. SCOTT evidently belongs to this small and select band, and we hope that he will have many more opportunities of using his gift for the advancement of knowledge and the increase of prosperity to mankind.

Dr. SCOTT: With regard to the analysis of the fruit, this recorded investigation was practically finished in August and September of last year. In the first investigation, I got the Deputy Island Chemist out there—who is an excellent man—to make the analysis. Thinking there was so much fat in it, he made it with alcohol, with ether, with petroleum ether, but so far as one could judge, such extracts were inert on the laboratory animals which were experimented with. And I think the mere fact of using these alcoholic things did not extract them. I said if you catch a child in the early stage of the condition, give it as much alcohol as it will take, as it is likely to precipitate the poison, and no secondary symptoms occur. I think in that way the chemist was

missing out the toxic principle. When making the soups with boiling water, the poison is practically extracted, although there is this fat on the top. It comes down even on filtering.

With regard to the question of the poison being a volatile substance, cassava is a case in point. The native will not take it until it has been washed three times in order to get rid of the prussic acid. The solid part was not administered with the liquid in these experiments, because I found that the most fatal cases were those of children who were given the soup or "pot-water." The older people, as a rule, after boiling, took out the solid part and ate that. Some of the adults who suffered severely did so, I believe, because they had some of the soup with the more solid portion. The older children who were given some solid and some soup were very ill indeed, and most of them died. The babies who had the soup alone invariably died. Therefore I cannot answer the question as to the solid being possibly an antidote to the extract.

The reason why the liver is so greatly affected I am afraid I cannot say. If the patient has died some time after the symptoms set in, all the organs—heart, pancreas, even the lungs—shew this fatty condition; the same is true of the lymph glands, as in the specimen I shew you. I think the poison, being in the stomach, is absorbed straight away through the vessels. It probably gets to the liver first, and thence passes into the general circulation. If you look at the cerebral sections you will see there are occasional drops of this staining with osmic acid in the smaller vessels, as well as, but less marked than, in the big nerve cells themselves. It looks as if the liver made an attempt to stop it—which agrees with the calm interval—and that it then got beyond that organ, and that it is when it reaches the brain that most of the symptoms are evident. I have found fat even in the heart muscle in these cases.

I cannot answer Dr. Low's question as to why the unopened fruit is toxic: that is the direction the further investigation will take. The natives know the fact themselves. I shewed them several fruits, and asked, "Would you eat this?" "No." "Why?" "I don't know." I think it may sometimes be due to it not being quite ripe. It is usually those with a small seed, the fruit itself not having properly developed. It is not hydrocyanic acid, because that would be given off in boiling. It is very difficult to get the history of a case from the native, as he fears being had up for poisoning his child: he would have to admit having

given his child something from which it died. I only know of one white person having had the disease at all, and he said he had not used the fruit from his own tree; he sent down to the market and bought a lot, and ate them with some friends who had come to see him. The others were new comers and did not like them, but he took them as he said he was fond of them. He nearly died; he vomited for twenty-four hours without ceasing. I found that a decayed fruit is not poisonous in that way. I left one to decay, and tried the effect of extract from that, and it was not poisonous. I have myself eaten the ripe fruit, when I have seen it gathered. It is more of a vegetable than a fruit, and is commonly eaten with fish. White persons throw away the water, using only the solid. It is boiled in a linen bag. The fatty degeneration in this disease is more acute than in phosphorus poisoning. It opens up the question whether some of the cases reported as yellow fever in West Africa may not be due to the eating of this fruit.

In answer to the Chairman, it would be practically impossible to cut down all these trees in the island, as they are so plentiful. Moreover, they blossom at a time when other kinds of fruit are very scarce. Now that pamphlets have been sent round warning the people of the dangers, there have been only a few cases during the whole of last year on the island, and, as far as I know, only three deaths.

ERRATUM.

In the List of References given after Dr. BAYON'S Paper in the December (1916) number of the TRANSACTIONS, Vol. X., No. 2, p. 32, an error has crept in. The 3rd reference should read, "Insect Flagellates and the Evolution of Disease, with Remarks on the Importance of Comparative Methods in Protozoology." *Annals of Tropical Medicine and Parasitology*, Vol. IX., pp. 335-347.

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NOTES ON TWO DISEASES OBSERVED IN KARONGA, NYASALAND, DURING THE PERIOD 1914-1916.

BY
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Shortly after my arrival in Karonga in April, 1914, a woman consulted me with signs and symptoms of a disease I had never seen before. Since then I have seen numerous cases of the same type. I was able to keep in touch with the woman until I left Karonga in July, 1916, and as her case is characteristic, I shall give my notes of it in full.

Case I.—Elizabeth, aged about 30, married, with two children, was first seen in May, 1914. She had married a second time a year before, had had no child by her second husband, and wished medicine to produce one. Nothing abnormal was found on vaginal examination; but the abdomen was nearly filled by an enormously enlarged liver and spleen. The photograph gives a picture of their size a year later. When I first saw her, liver and spleen were even larger. The spleen then came to within an inch of Poupart's ligament. Blood examination, frequently repeated

since, has never shewn parasites, although the blood elements, to be referred to later, are abnormal. The stool contained ankylostome ova. After one course of treatment I could never again find any. Most people in this neighbourhood harbour a few ankylostomes. Her illness was certainly not due to them.

The patient had walked three miles to see me, and, though she did not feel strong, had kept at work both in house and field. Vague symptoms had begun, insidiously, about a year before. (In such a place as Karonga the time of onset of a disease is rarely recognised. Sub-acute infections are so frequent, and chronic infections, fevers, dysenteries, diarrhœa, are so universal, that a degree of ill-health is scarcely considered abnormal). Her symptoms were irregular chills, flushes and sweats, progressive weakness and shortness of breath, and progressive abdominal distension.

Beyond enlargement of the liver and spleen, there were few physical signs. The abdominal wall was extremely flaccid and thin, permitting the hand to dip underneath the liver and the spleen and to feel the gall bladder and the outline of the colon. Mucous membranes were pale and livid. During May and June, 1914, her morning temperatures—when I had the opportunity to take them—were between 96° and 100°, and her evening temperatures between 98° and 103°. I gave full doses of quinine, and, later, arsenic, without effect.

I went to Kota in July, and returned to Karonga with the Field Force in August. In September the patient came to me with the news that she was pregnant. She seemed a little better and believed her pregnancy due to my treatment. In the seventh month of pregnancy she was dangerously ill with amœbic dysentery; but the child was born at full term, quickly, without interference and without maternal complication. The child was suckled by the mother, has been healthy but for several attacks of malaria and one of dysentery—few infants escape either in these parts—shews no sign of her mother's disease, and in July, 1916, was fourteen months old and alive and well.

During these two years I have seen the case frequently. Her abdominal condition shews no temporary variations. I have been unable to take daily temperatures, but she has pretty constant irregular fever, her evening temperature rarely being normal. Twice she has taken

30 grains of quinine daily for more than a week, without effect. I have never found a single parasite in at least twenty examinations. Arsenic by the mouth and soamin intramuscularly had no observable effect. During the two years there has been irregular but perceptible improvement in health. The lower edge of the spleen has retreated about three inches, and the liver edge—at one time four inches beyond the costal margin—is now only two inches beyond it.

During my stay in Karonga, I have had five cases similar to this in hospital, and have seen many others amongst out-patients.

I shall now give a summary of the distribution of the disease and of its signs and symptoms, and conclude the account by a description of the only case I have been able to watch from the onset of symptoms.

The disease is one of later childhood, adolescence and early middle age. I have not seen a case in an infant, nor in any one over forty. Cases have come to me from all round Karonga, but there is a special focus of the disease in the villages on the banks of the Rukuru. I collected over a dozen cases in children aged 5 to 15, from within a half-mile radius, two miles up the Rukuru from Karonga. So far as I can judge, most cases die. I know of no death in under two years from the onset of symptoms. I have seen several cases which I believed to be certain to recover, the one previously narrated being one of these. One boy, with the usual enormous liver and spleen, has been constantly at work in the Regimental Store of the King's African Rifles for the last eighteen months.

The onset of the disease is generally insidious in nature. Some histories suggest that the real onset is preceded by several ephemeral attacks of fever. Throughout the course of the disease fever is almost constant, with morning remissions. Weakness, anæmia, and abdominal enlargement come slowly. When fully developed, the enlargement is very characteristic, especially in children. The lower ribs and upper abdomen bulge out on both sides, allowing cases to be picked out by the eye in a crowd of naked villagers. Natives call the disease "wenku" (Kinkonde) meaning spleen. They confuse it with malaria, but say that when the lump is on the right as well as on the left side of the abdomen the case will probably die. They say that all children who get the very swollen abdomen eventually

die. They believe that the disease has been introduced since the arrival of Europeans into the country.

The liver is not always enlarged. I have seen both advanced and early cases with no liver enlargement; but when splenic enlargement is not excessive, and when it is not accompanied by hepatic enlargement, it is impossible, without repeated blood examinations, and without watching the results of quinine, to be sure cases are not malarial. In one village, out of 69 children, 67 had enlarged spleens. Of the infants none had markedly enlarged livers. The eight children with enlarged livers were all over four years old. In other nine the degree and direction of splenic enlargement were against the diagnosis of malaria. Malarial spleens that I have seen rarely pass far beyond the umbilicus. In the present disease the enlargements of liver and spleen are characteristic in shape and direction. The spleen (*vide* photographs) enlarges in the direction of the spine of the ilium—I have in two cases been unable to get my fingers between the organ and Poupart's ligament. The liver, while enlarged in all directions, is often most prominent near the middle line. The left lobe is then separately palpable and often makes a special prominence below the xiphoid, visible to the eye. (My only two photographs unfortunately do not shew this special feature). These directions of the enlarged organs are, I presume, due to their pressure upon one another. Both liver and spleen are usually firm and smooth on palpation; but in one case, convalescent when I saw him, both organs were remarkably hard. The spleen, which extended to four inches beyond the ribs, was smooth. The liver, felt two and a half inches beyond the ribs, was very hard, and apparently entirely composed of large nodules. This man complained of liver pain—the only case I have seen who did. He had a history of many years' illness, but said he had recovered, and, except for his abdominal condition, looked and was perfectly well.

A thin and flaccid abdominal wall is a notable feature of the disease. Peristalsis is often visible. Attachments of the liver and spleen are so loose as to permit both to move forwards and downwards in the erect as contrasted with the recumbent posture. In the case of the spleen the edge may thus move as much as four inches.

Many children with the disease get very thin, but adults are always well covered. General œdema develops for some time before death.

None of my cases died in hospital, so I have been unable to do post-mortem examinations.

The disease has a characteristic and very uniform blood picture. No parasites beyond once—a filarial embryo, and several times a malarial ring—have ever been seen. I once punctured a spleen, twice punctured a liver, and twice examined the fluid of a blister without finding anything recognisable as Leishman's bodies. Eight blood examinations of Case I., extending over two years, gave the following variations:—

Large mononuclears varied from	31	per cent.	to	49	per cent.
Lymphocytes	9	,,	to	20	,,
Eosinophiles	0	,,	to	10	,,
Polymorphs	32	,,	to	46	,,

Many of the polymorphs were immature. The smallest proportion of large mononuclears and the largest of polymorphs were seen just after the birth of the child. The child's blood was twice examined. Once the large mononuclears were 22 per cent. Subtertian rings were then present. A month later they were 12 per cent. I have seen no case of the disease where the large mononuclear count was under 27 per cent. Many of these are always of the large lymphocyte type, some very large, with scanty deeply staining cytoplasm. Cells of that type, in these cases, so grade into ordinary hyaline mononuclears, as to make distinction difficult. A degree of lymphocytosis is always present. I have been unable to do estimations of red and white cells, or of hæmoglobin. Among members of the Field Force, which, until late in 1915, had an average enlisted strength of over a thousand men, one case of the disease occurred. That other cases occurred and were missed is quite impossible. This man, Moffat, a stretcher-bearer, came from Zomba with the troops in August, 1914, and was practically continuously resident in Karonga until he took ill in July, 1915. He was admitted to hospital on July, 14th, on the fifth day of his absence from work, although he had been feeling unwell for months previously. The character of the fever can be seen from the temperature chart, which I enclose. On admission the spleen was just palpable. For the first month the temperature was 103° to 105° in the evenings, 99° to 103° in the mornings, pulse rate 90 to 100. There were some bone pains. There was no albuminuria. The chest was, and remained, clear. The spleen continued to enlarge,

and in November, three months after admission, was at its maximum, the edge an inch above the umbilical level. The liver began to enlarge early in August and reached its largest at the same time as the spleen, then extending from the fifth rib to two and a quarter inches beyond the ribs in the nipple line, curving thence slightly downwards to the middle line. At that time the lower chest and upper abdomen were markedly distended. When he was sent home to Zomba in January the liver was undiminished in size. The spleen had slightly diminished. During October there was cardiac weakness with a faint first sound, a pulse rate of 110 to 130, and œdema of the legs. From August to November there was complaint of pain, in localised areas, mainly in the upper arms, less often in the thighs. These pains came and went as the evening temperature rose and fell, were referred to bone and lasted from one to five days in one place. The chart shews a very slow fall of temperature from the first month. During November it first reached normal, and the patient appeared to be making a final recovery, but the temperature rose again to 101° in the evenings. In December it was almost steadily normal, but the fever returned during the fortnight before discharge on January 5th, 1916. In October there was slight albuminuria. There never were any abdominal symptoms, and the tongue was clean throughout. By October the patient had lost much weight, but never became emaciated, and before the temperature had reached normal was gaining weight rapidly, and, in spite of the persistence of hepatic and splenic enlargement, had got quite fat when he left hospital. Except for one week in October the patient used to sit outside in the daytime, went by himself to the latrine some twenty yards away, and throughout the course of the illness the colour of mucous membranes was fairly well preserved. His blood counts were as follows:—

	Aug. 17.	Oct. 10.	Oct. 25.	Jan. 2, 1915.
Large mononuclears	30 %	36 %	44 %	25 %
Lymphocytes	19 %	16 %	19 %	21 %
Eosinophiles	5 %	3 %	2 %	2 %
Polymorphs	46 %	44 %	35 %	52 %

No malarial parasites were ever seen. Stool examinations for ova and amœbæ were negative. I thought the case one of enteric at first; but Widal's reaction (dead cultures) was negative in August and October.

And the cleanness of the tongue, the absence of emaciation, the length of the course of the disease and the blood picture, to my mind exclude the diagnosis of enteric.

I shewed cases of the disease to all the Medical Officers with the Field Force at the time, and they all recognised it as having uniform and specific characters. I owe the two photographs to Major STANNUS, the Deputy-Director of Medical Services, who also photographed many other cases which I shewed him. One of the photographs I enclose is that of Case I. The other is of a rather early case.

In CASTELLANI and CHALMERS's book on Tropical Medicine the disease is described as tropical splenomegaly. They describe two stages, in the earlier of which there is no hepatic enlargement. In many of my cases the liver enlarged together with the spleen. In some advanced cases hepatic enlargement was slight or absent, and in all, though there were frequently remissions and exacerbations of the disease before its course settled down to recovery or death, the characteristic signs did not vary. Accounts of the same disease have appeared in the *Bulletin of Tropical Diseases*. But in all the accounts I have seen the disease is referred to as being comparatively rarely met with. Around Karonga it is certainly common. Dr. INNES, of Livingstonia, recently shewed me notes of what were certainly cases of the disease, seen when he lived in Karonga twelve years ago. He used to think them intractable malaria, and his recollection of them is that they all eventually died. All his cases were in adults.

The disease I have described I am sure I have never seen, in fifteen years of East Africa and Nyasaland, outside the North Nyasa district.

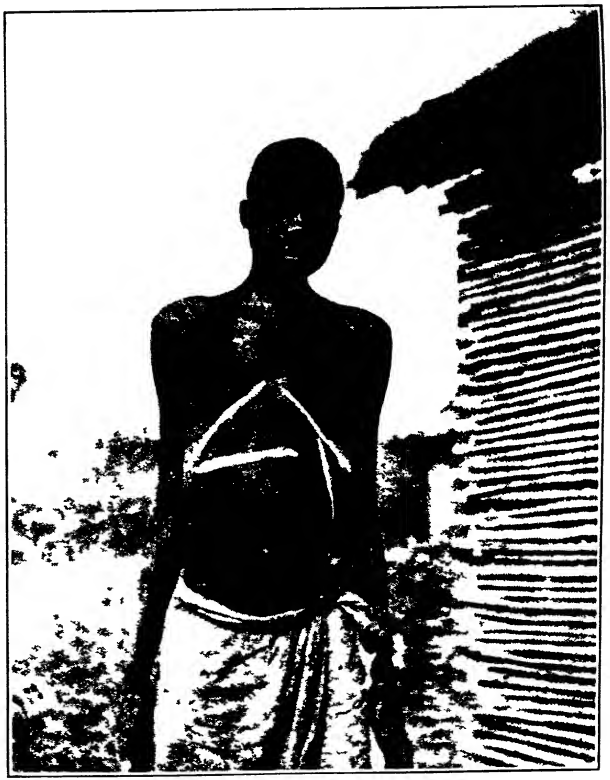
The next disease I am about to mention, although I know of no published account of it, I have seen before. I used to see isolated cases at intervals of years, wonder what they were, and forget them. I may define the disease as multiple non-suppurative swellings of connective tissue. The swelling may occur literally anywhere in connective tissue, inside joints, under periosteum, in muscle (the commonest site of all), or just under the skin. They may be single but are more often multiple. When multiple they may arise and resolve together or they may succeed one another, often overlapping in point of time. Some of the first cases I saw suppurated. That result I now attribute to mistaken treatment, particularly the use of liniments or fomentations. After I confined

treatment to the immobilising of the affected part, none of the swellings suppurated. I shall mention a few illustrative cases. In August, 1914, a porter came to the Dispensary at Karonga, with a large, firm, rather diffuse and slightly tender swelling, which had begun to arise several days before, apparently situated under the fascia of the outer side of the thigh. I left it alone—it was not the first case I had recognised—and it disappeared in a few days. In August, 1915, an askari was sent to hospital with a swelling about the size of an egg over one of the lower ribs. It had been fomented for several days, and, just before admission, had been punctured with a hypodermic needle. Pus was found, and the case was sent to me for operation. For one day there was slight fever. I left the swelling alone, except for a bandage, and in nine days it had quite gone. As it was subsiding another swelling developed in the right calf. That one rose and fell in six days. The patient was back to duty in fourteen days after admission.

A porter came to hospital in September, 1914, with five swellings. Three were in arm muscles and one on the back of one of his shoulders. The fifth was in the left knee-joint, which was painful and greatly swollen. The patient would not let me use even a needle, so I tried Scott's dressing, with the result that pus developed under the epidermis, which peeled off. On using simply a splint and cotton wool he improved for a time, but after moving about, as natives will, he got worse. The swelling was extreme, pain was severe, and he became febrile in the evenings. The hospital was full of wounded at the time, and we were trying to evacuate as many of the other patients as possible, so I sent this man by steamer to the Mission Hospital at Livingstonia. I learned later that, after many weeks, all the swellings finally subsided without suppuration.

The most chronic case I had was in a porter who was in hospital for nine weeks. Both wrists and both ankles were swollen. The dorsum of one hand (for a week only), and an area of about six inches square in the lumbar muscles were also affected. For six weeks he lay perfectly helpless. But for the swelling in the back one might have used the word rheumatism. He never had fever and the pain was never severe. Improvement when once begun was very rapid, and on his discharge there was nothing to shew but some stiffness of wrists and ankles. I have seen one fatal case. In June, 1916, when I was at Ngara, a houseboy

CASE I —ELIZABETH



CASE II —AN EARLY CASE OF THE DISEASE

Illustrations accompanying Dr Norman Leys Paper

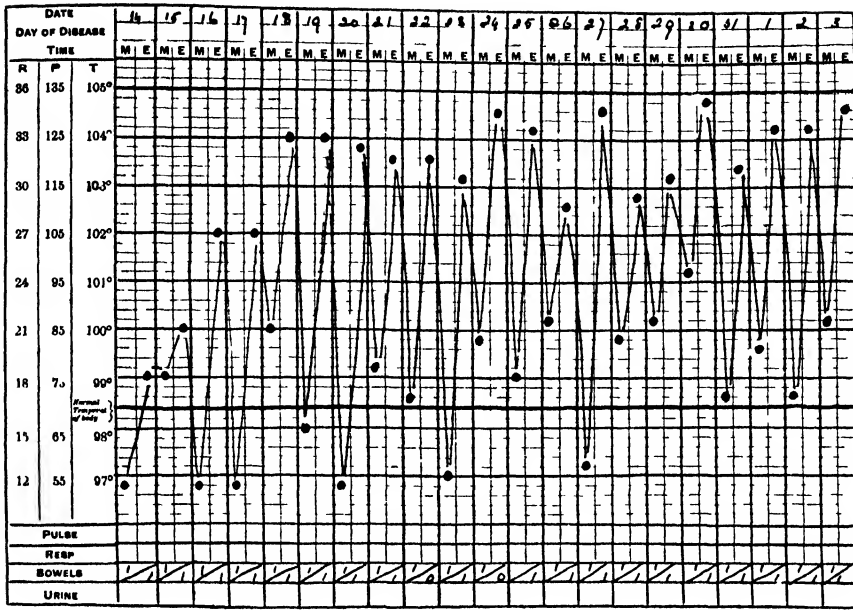
came to me with pain referred to the right hip. There was nothing to feel and nothing to see beyond slight lameness. The next day he was so lame as to be unable to walk, although there was nothing visibly or palpably wrong. On the previous evening he had developed a diffuse swelling over the left scapula. The pain there was slight. There was no local heat and no fever. I gave him Epsom salts and ten grains of aspirin to be taken later. When I went to see him the next morning I found he had died in the night. He had been choked by a swelling of the pharynx and palate on both sides. The shoulder was still visibly swollen after death. I have seen no other case where the swelling occurred under the mucous membrane. Natives know the disease and call it "vimba," which means swelling; but they call other diseases "vimba" too. They counter-irritate, which does harm. The one essential of treatment is to immobilise. Active treatment is harmful, and the internal remedies I have tried—mercury, iodide, arsenic, sodium salicylate—are useless. Pain is only considerable in some joint cases. A few cases have slight fever at first. My earlier cases I used sometimes to incise. When I got pus, as sometimes happened, smears shewed no organisms. More often tissues were found simply œdematous. In one case there was fluid in a tendon sheath. I repeatedly punctured swellings with a hypodermic needle without finding any organisms. Once I dropped a few drops of blood from a swelling into a test-tube containing sterilised broth. Nothing grew. The most natural theory of these cases is that they are due to an attenuated virus carried by the blood, a virus causing inflammation short of pus formation. For long I suspected they were filarial. I am now sure that they are not. I cannot profess to have examined the nocturnal blood of every case. But I examined the blood of most and only found embryos in one case, and they were scanty.

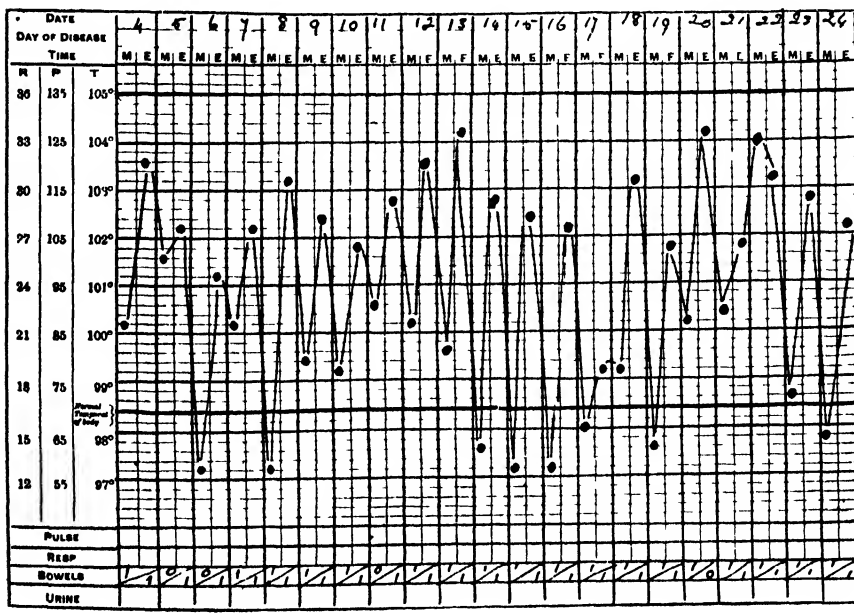
In November, 1915, during routine stool examinations, I first saw an organism which I cannot find mentioned in CASTELLANI and CHALMERS. It is an oval organism, about as long as the diameter of a red blood corpuscle, and so active as to prevent observation of its structure before it dies. After death one or more vacuoles develop. It was unaffected by stains run on to the slide. In fixed and stained slides, specimens were only visible as faintly stained outlines not very distinct. They were so abundant in some stools, during November and December, 1915, that more than a dozen specimens were visible in a

field of a one-sixth inch objective. For these two months the parasite was common in stools, but after the end of December they were rare. They were abundant in the stools of one European, a South African soldier, with amœbic dysentery. When patients without dysentery had these parasites they always suffered from a chronic not very severe diarrhœa—they usually had from two to six loose frothy stools in the twenty-four hours. I thought thymol did good, but sometimes diarrhœa and parasites persisted for weeks. They all cleared up in time. I sent at least two men back to duty with these organisms still present in stools of practically normal consistency, and they did not return to hospital.

MOFFAT'S TEMPERATURE CHARTS

JULY, 1915 AUG.





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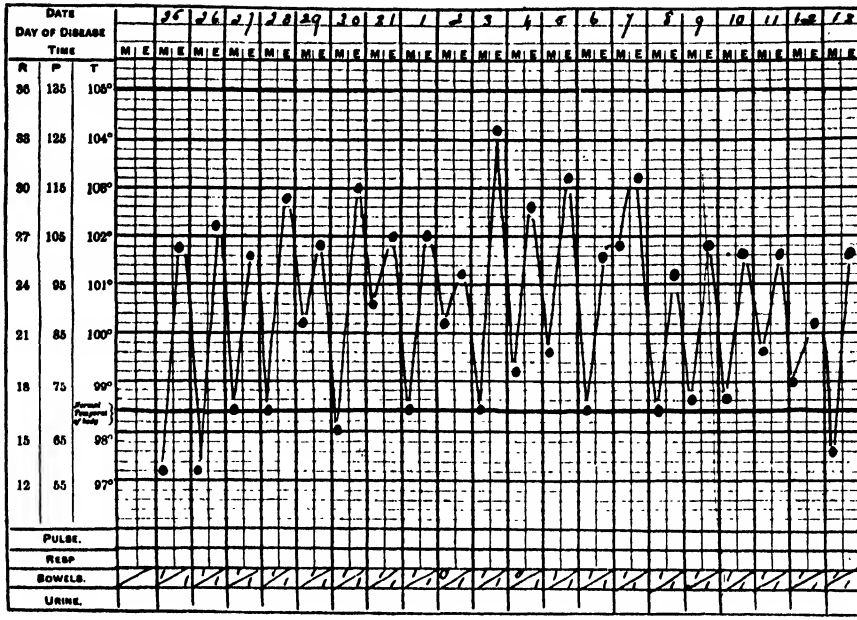
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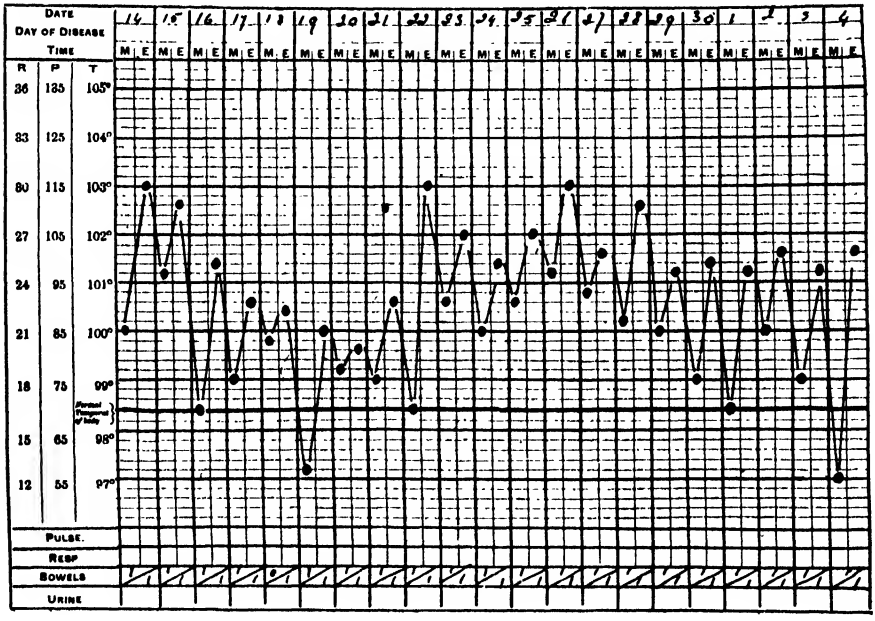
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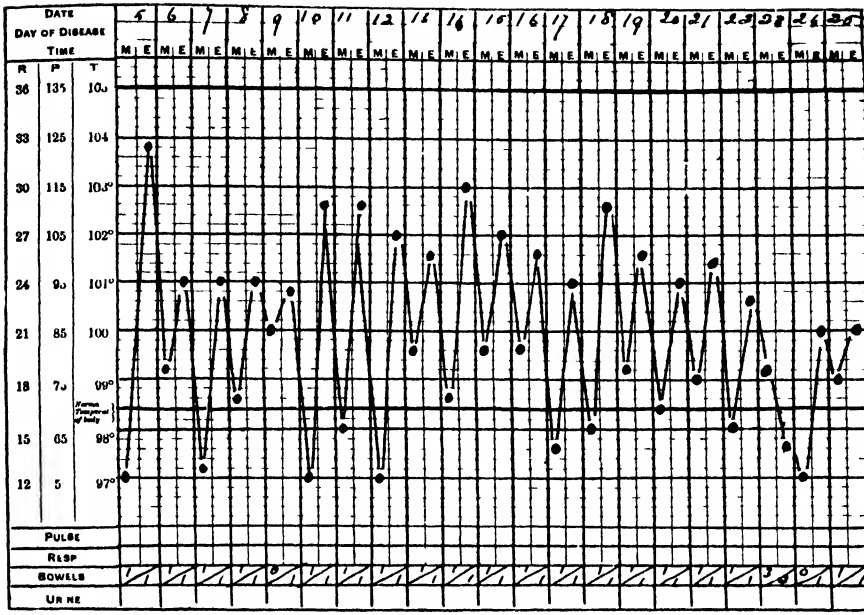
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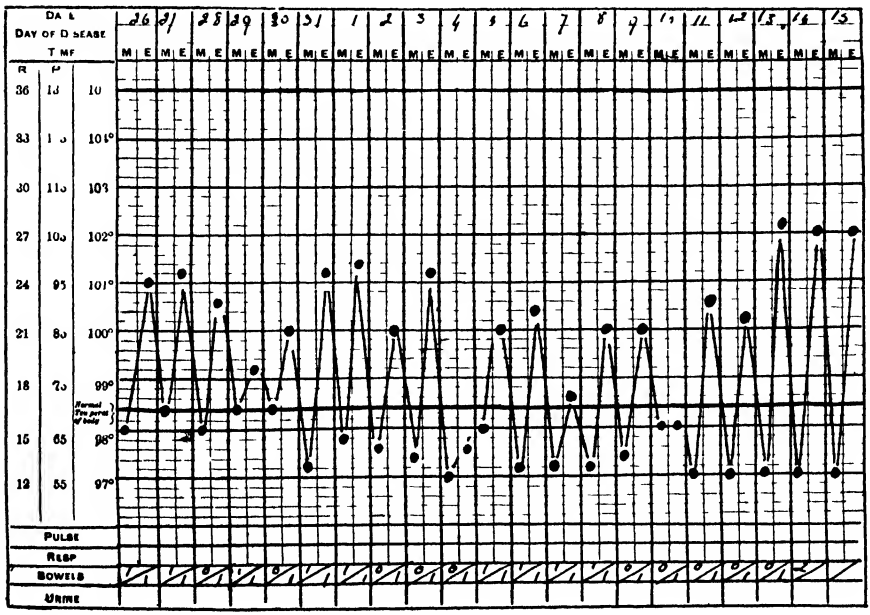
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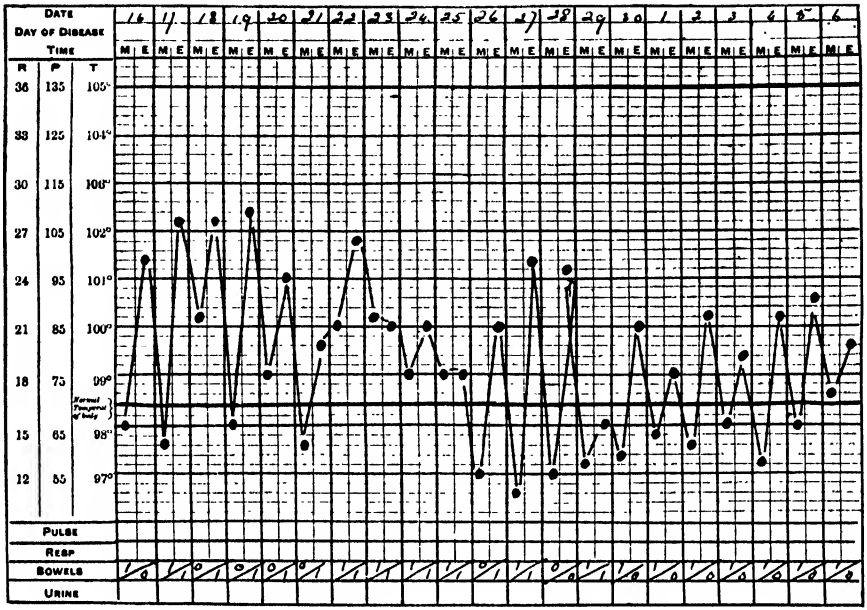
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Normal Temperature of body
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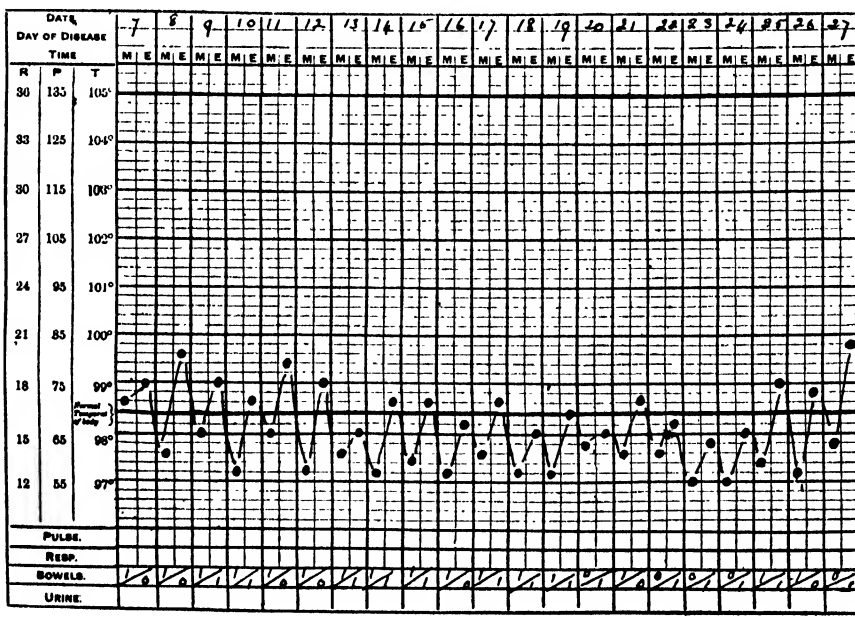
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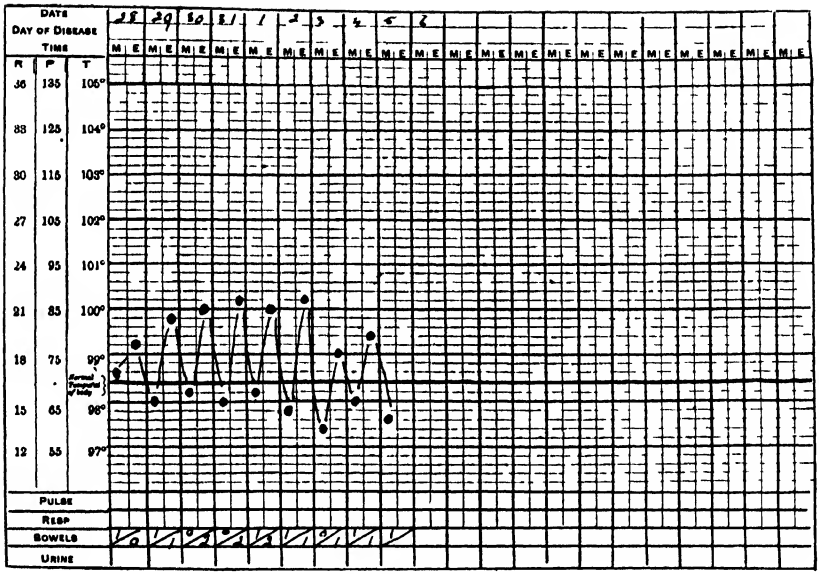
JAN., 1916.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FIVE HUNDRED AND FORTY-TWO CASES OF YAWS TREATED BY KHARSIVAN AND ARSENOBILLON.

BY

P. HARPER, M.R.C.S., *District Medical Officer, Fiji.*

Five hundred and forty-two cases of yaws were treated by the writer during the half-year May to October, 1916. All these cases received kharsivan and arsenobillon, 445 being treated by injection into the veins and 87 by intramuscular injection. Eighty-six of these cases received two or more injections. The treatment was carried out in the Provincial Hospital at Ra, with the assistance of one native medical practitioner and three native dressers from neighbouring villages. Pure sodium chloride and pure sodium hydroxide were used for most of the cases, but for a minority it was necessary to make use of commercial salt and commercial caustic soda. Rainwater was used instead of distilled water in all cases. There were no severe reactions.

Two cases suffered from bad after-effects. One of these was a male adult Fijian with multiple periostitis, involving chiefly the bones of the limbs and head, and with such severe headache as to make him quite useless for all work and almost incapable of conversation. In this case the cephalic vein, into which the injection had been made, had to be excised for gangrene. Healing was rapid and uneventful. No unpleasant symptoms followed his second injection. The man left hospital completely cured of all his symptoms.

The second case was also a phlebitis, but much milder. The patient was a Fijian clerk suffering from rheumatic pains due to yaws. He received the ordinary intravenous injection of 0·4 gram of kharsivan, this being followed by a sharp attack of phlebitis, which yielded rapidly to laxatives and evaporating lead lotions.

In my opinion, both these bad results were caused by giving the kharsivan in too strong a solution.

The only case not deriving any benefit from the injections was a case of G.P.I., who ran away shortly after his first injection. One doubtful case of G.P.I. has been lost sight of after considerable improvement in her condition. The third case of G.P.I. has had four injections of 0·4 gram each intravenously, and his symptoms have slightly ameliorated.

In all other cases there was a definite cure of all symptoms of active yaws.

Can it be proved that salvarsan and its substitutes really cure yaws? On August 7th, 1911, the writer treated by intravenous injection of salvarsan an Indian child with very extensive secondary yaws. On September 5th, 1911, he treated by intravenous injection, followed some days later by intramuscular injection, of salvarsan a male European, aged fifty-seven years, who was suffering from advanced tertiary yaws, ulceration and cachexia. These cases were referred to in a previous paper to the *Lancet* (August 8th, 1914, page 370). In the case of both these persons, and in the case of a half-caste woman referred to as case No. 7 in that communication, the cure is known to have remained complete for a period considerably over five years. Several others of my original cases have been traced for shorter periods of time and none of them have recurred.

The old German salvarsan, then, seems to have been an absolute cure for secondary and tertiary yaws. The recent substitutes seem to the writer to be weaker, but quite efficient in increased doses, and not nearly so toxic.

The table given below summarises the chief manifestations of yaws in these cases.

The case of primary yaws was interesting because it was undoubtedly

a case of accidental, not purposive, infection. All purposive inoculations by the Fijians are done after the eruption of the milk teeth. This child was toothless. The parents were unable to say where she contracted her infection, but she was probably infected by her elder sister, who was suffering from extensive secondary yaws. The site of infection was the sole of the left foot, and the necrotic area was the size of a half-crown, with an ulcer in its middle the size of a sixpenny bit. The case was cured within a week by the injection intramuscularly of 0·05 gram of kharsivan.

In the table given below the cases of tertiary yaws are tabulated to show the most common manifestations. With Fijians there can be no question of mistakes in diagnosis, because at present the Fijians are quite free from syphilis, whilst they all suffer from yaws.

A few words of explanation may be needed as regards some of these manifestations of tertiary yaws.

The "soki" or crab-yaw, is an exceedingly painful affection characterised by a granulomatous mass slowly ulcerating its way from the subcutaneous tissues through the skin of the sole or palm to the surface. The granuloma may be sessile or stalked, and its base or stalk is often bathed in a quantity of greyish stinking pus. As a sequel to this condition of soki or crab-yaw, especially if the sokis have been situated near the metatarso-phalangeal joints, hammertoe of one or more digits may develop. The Fijians, of course, do not wear boots.

"*Kakaca*" *or* "*vula*" is a superficial dry dermatitis, the nature of which is shewn by the following description of a typical case :—

No. 367.—Hands : The palm of the right hand from the wrist to the finger-tips is red, shiny and fissured. The dorsal surface is healthy. The palm of the left hand is red, shiny, fissured, and pitted from the wrist to the finger-tips. Just above the wrist, on the flexor surface of the left forearm, there are a few patches of depigmented skin, the bright white centres of which gradually fade into the normal brown of the skin around The dorsal surface of the left hand and fingers shews patches of white atrophic skin without signs of active disease. The patient says that these white patches are where the kakaca has healed—that it is the end-result or natural cure of the condition of desquamation, pitting and fissuring mentioned above.

Feet: The softer skin of the instep and toes is shiny and reddened, and has branny scales of desquamation on it. The hard skin of the heel, sole and ball of the feet is very thick, of a dull yellow colour, and is deeply fissured in various directions. On the dorsal surfaces of both feet the skin is affected with a psoriasis-like rash, with fine silvery scales of desquamation. The skin between the toes is also affected, and here the margin of the affection is indefinite. In an upward direction the margin is clearly defined from the surrounding skin. The margin is everywhere made up of little coalescing circles of desquamation. On the left side, where the affection is obviously less advanced than the right, the affection consists almost entirely of such little circles of desquamation. On September 1st, 1916, this patient received an intravenous injection of 0·4 gram of kharsivan, and on the 6th the affection was noted as much decreased in extent and severity. The patient was discharged cured on the 13th.

In this affection, then, the first change is a desquamation over a number of very small areas of skin. Then follows pitting and fissuring of the skin, then loss of natural pigmentation with often a bright red or beautiful "flesh-pink" colour. The final stage is a dead-white atrophic area, often mistaken by laymen for leprosy. The relative amount of splitting, fissuring or pitting seems to depend on the hardness of the skin, these conditions being much more marked on the hard plantar or palmar surfaces, whereas on the softer skin the condition may pass from desquamation to depigmentation without any pitting or fissuring being noticeable. From the table this kakaca will be seen to be one of the commonest manifestations of tertiary yaws. It is usually confined to the palms and soles, but sometimes extends up the limbs, and I have seen it affecting the lips and chin. When affecting the palms it is a frequent cause of Dupuytren's contraction.

Rheumatism—or perhaps rheumatics would be a better name—is also a common manifestation of tertiary yaws. The patients with this complaint may or may not shew objective signs of yaws, such as periostitis or synovitis. Usually I have found no such objective signs, though scars of various sorts are common. The complaint of the Fijian patient is almost invariably as follows: "When the sky becomes dark my whole

body aches," or, "When rain or a cold wind comes I have to lie down, and am no use for work." This rheumatism can be relieved by potassium iodide and cured by salvarsan.

Under the heading, "Other manifestations," in the table, are included two cases of deafness, which were certainly in part due to periostitis of the bony meatus of the ears. Both these cases were very considerably improved by treatment, their hearing on discharge being almost normal. Under this heading also are included two cases of sciatica and one case of painful thickening of the skin of the ball of the foot. This latter condition is rare in Fiji. It yields readily to treatment with salvarsan or its substitutes. Two cases of reported abortions or still-births by Fijian women were treated by kharsivan, and both subsequently bore healthy full-term children. One of these two women was also suffering from periostitis of the tibia. It will be noticed that there is no mention of cardiac or arterial disease amongst these cases. This is a coincidence, as arterial disease is a fairly common result of yaws.

TABLE SHEWING SYMPTOMS OF YAWS IN 542 CASES TREATED BY KHARSIVAN AND ARSENOBILLON, AT THE PROVINCIAL HOSPITAL, RA, FIJI, MAY TO OCTOBER, 1916.

Aged	Sex.		Total Cases.	Primary Yaws.	TERTIARY YAWS.		GUMMATOUS ULCERAT'N							PERIOSTITIS													Tabes & G.P.I. Paraframbœsial dis.	Total manifestations of yaws, I., II., III. & IV.
					Secondary Yaws.	Of pharynx and larynx.	Of face and head.	Of trunk and scrotum.	Of breast.	Of limbs.	"Sokia" or crab-yaws.	"Kakaca" or "vula," superficial dry dermatitis chiefly of flexor surfaces of feet and hands.	Hammer toe and Dupuytren's contraction.	Of phalanges, metacarpals, metatarsals, carpals, tarsals.	Of bones of face and head.	Of long bones of upper limb.	Of long bones of lower limb.	Of shafts of ribs.	Synovitis.	Adenitis.	Rheumatism.	Transverse myelitis.	Paralysis of cranial nerves.	Cerebral yaws (coma, etc.).	Other manifestations.		
	M.	F.																									
7 months .		1	1	1		—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1
From 7 mos. to 3 years	6	6	12	—	10	—	—	—	—	—	1	—	—	—	—	—	1	—	—	—	—	—	—	—	—	—	12
From 3 yrs. to 10 yrs.	27	25	52	—	16	—	2	—	—	1	29	3	—	3	—	1	7	—	—	—	1	—	—	—	—	—	63
10 yrs. and up to 20 yrs.	49	23	72	—	1	—	2	—	—	2	51	5	1	—	—	—	5	—	—	—	13	—	-	—	2	—	82
20 yrs. and up to 40 yrs.	135	78	213	—	—	3	3	2	1	19	91	71	8	2	4	2	25	—	3	1	61	1	1	—	7	1	306
40 yrs. and over	119	73	192	—	—	3	2	2	1	15	55	79	18	1	4	3	24	1	2	—	85	1	2	1	9	2	310
All ages ...	336	206	542	1	27	6	9	4	2	37	227	158	27	6	8	6	62	1	5	1	160	2	3	1	18	3	774

NOTES AND COMMENTS.

THE HISTORY OF THE USE OF INTRAVENOUS INJECTIONS OF TARTAR EMETIC *(ANTIMONIUM TARTARATUM)* IN TROPICAL MEDICINE.

Dr. C. W. DANIELS has pointed out to me that my remarks as regards the use of intravenous injections of antimony for trypanosomiasis at the London School of Tropical Medicine are not quite correct. He proposes that in the last paragraph on page 38, four lines from the bottom, "some" should be written instead of "most," and that in the last line of the same paragraph "my" should appear before "standard."

The corrected paragraph would therefore read as follows:—"Since that date a combined treatment by atoxyl and intravenous antimony (tartar emetic) has been carried out on some of the human trypanosome cases that have passed through the London School of Tropical Medicine, and such treatment has been my standard one for this disease for many years."

G. C. LOW.

TRANSACTIONS
OF THE
SOCIETY OF TROPICAL MEDICINE
AND HYGIENE.

MARCH, 1917.
VOLUME X. No. 5.

SOME OBSERVATIONS ON BRAZILIAN BERIBERI.

BY
JOHN W. LINDSAY, M.B., LIEUT. R.A.M.C.,
BELÉN, PARAGUAY.

Recently in conversation with a Brazilian doctor, Inspector of Ports for his Government, I asked him regarding the etiology of beriberi. He replied that the disease is due to the deficiency or absence of vitamines in diet, and especially in rice, so much so that steps were being taken to prohibit the importation of polished rice into the country. Questioned as to the disease known as beriberi in Matto Grosso and the Amazons, he replied that it is quite a different disease, being a "quinine-resisting form of paludism." Whether or not this represents the official theory of the Brazilian authorities I have not been able to ascertain. My own observations, however, and information obtained from various other sources, may be of sufficient interest as to merit consideration from those whose special work it is to investigate the etiology of such diseases.

I made special enquiries regarding the disease in the towns and districts of Corumba, and Cuyaba, the capital of the province. A Brazilian colleague, Dr. HORACIO HUIQUE, Medical Officer of the Military Hospital

of Corumba, sent me detailed information regarding the disease in his district:—

"Geographical distribution: the city of Corumba and neighbouring districts extending towards the Amazons and Bolivia.

"Locality: it occurs in low-lying districts, near lagoons, and along the course of and in the neighbourhood of rivers.

"The soil: the nature of the soil varies, but it is always in places liable to periodical inundations, or in places in the neighbourhood of swamps, even although not liable to inundation.

"The season of the year: mostly during the summer months, from November till March.

"The age and sex of patients: cases seen have been only in men, their ages being from 20 to 45.

"The occupation of the patients: they have been soldiers and sailors—soldiers who lived and fed in the barracks and were attacked there; sailors of the flotilla at Ladosio, who were attacked especially when living on board ship anchored at the riverside, it being necessary on more than one occasion to disembark the whole ship's company in order to fight effectually the epidemic.

"Conditions predisposing to the disease: overcrowding and poverty.

"Symptoms: generally the disease begins with fever; but the most constant initial symptoms mentioned by all the patients are pains in the lower limbs, numbness and powerlessness of the legs, and fatigue on the slightest exertion. There may be gastric disturbances. The symptoms of dropsy, loss of voice, and paralysis may appear very quickly, even within forty-eight hours in grave cases, or may develop more slowly in milder cases.

"Transmission of the disease: as regards insects, the only insect that seems constantly found in places attacked by beriberi is the mosquito Anopheles.

"The common food: rice, either imported or native grown and prepared, wheaten flour, flour of mandioca, meat (fresh or dried), and beans.

"Endemic districts: Corumba, Miranda, and many other special localities.

"The effect of change of climate: near Corumba, at a distance of twelve miles, is Urucum, which is high, bracing, and very dry, and the chief resort for beriberi patients from Corumba. The south of the State

and also the plateau of Campo Grande to the north-east have excellent climates for the cure of beriberi. It is worthy of note that all the places considered suitable for the cure of beriberi are at higher altitudes, and have dry bracing climates, the soil is dry and never subject to inundations, distant from swamps and from rivers with swampy margins."—(Report of Dr. HORACIO HUIQUE, Corumba.)

A beriberi patient from Matto Grosso gave me the following history. In 1895-96 he was living in Corumba, employed in a large business house in which there were thirteen employees in all. Ten of them were attacked with beriberi—at first two men, then one by one, until within a month ten of them had sickened. The chief symptoms were dropsy and paralysis. Some of the patients had to be carried on board the river-boats, others were able to walk down with the help of two sticks. They all went to higher altitudes and returned cured within two months, all except one who went to his home at Miranda, where beriberi is endemic, and died.

Their diet previous to their illness had consisted of meat, fowl, milk, white bread, vegetables, salads, fruit, native-grown rice, and maize. They drank the water of the river. The patients were all seen by the Brazilian doctor, Dr. ALEXANDER VALLE, who diagnosed the disease as beriberi.

The patient who related this story, Antonio P. Carvalho, had, after twenty years, a second attack of beriberi, and came under my care. Forty years old, a native of Cayuba, later resident at Corumba, where he had his first attack of beriberi, and now resident in the south-eastern corner of the State towards the basin of the Upper Parana, he fell ill on the 20th June, 1915, but was not seen by me until the beginning of August. His first symptoms were those of fever, and pains in the joints and epigastrium, numbness of the hands and feet, and swelling of the legs. Realising that he was seriously ill, he resolved to undertake the journey to the nearest Brazilian town of Punta Rora, and so, on the third day of his illness, he made his men carry him on a stretcher some fifteen miles to the house of a friend on the way to the town. Even this short journey was almost too much for him, and he had to remain there some three weeks. The fever lasted ten days in all, but the weakness and swelling continued. His legs were completely paralysed. He gradually

lost his voice, at first being unable to articulate the sounds of *r* and *l*, then gradually becoming completely aphonic. This aphonia lasted some eight or nine days and then his voice began to recover. He began to feel his feet and to be able to move them. He had become exceedingly weak and very anæmic. The improvement in his condition began from the time he left his friend's house, on the 16th July, and by the time he had covered the ninety miles to reach the town on the hills he was feeling remarkably well. Wishing, however, to make absolutely sure of a complete cure, he came on, 210 miles farther, to my place, arriving in a bullock cart on the 9th of August, fifty days after the beginning of his disease.

The patient was extremely emaciated and very anæmic. The left leg had begun to recover, and there was a certain amount of control over the foot. The knee reflex was completely absent. The right leg was completely paralysed and the knee-jerk absent. There was considerable numbness of the legs and hands. On several occasions, when trying to lift a glass to drink, he let it slip from his fingers. On one occasion, when he was feeling very much improved, in the absence of his servant he tried to take his medicine himself. He told me that he reached for the bottle, grasped it firmly—as he thought—by the neck, and raised it from the table, but it immediately slipped from his grasp and fell to the floor The voice had not completely recovered. There was a dilatation of the right side of the heart, with systolic murmurs. There was no albumin in the urine. The feet were still swollen. The knee-jerk of the left leg was the first to return, and that of the right leg a month later. The heart condition was so critical that he was not allowed to rise until the middle of November. During all the time he was in bed, his servant had massaged and moved his legs, so that when he was allowed to rise, he was able, within about a week to walk by himself. His gait, however, was for sometime slightly ataxic.

This patient's residence, where he had lived continuously for six months before he contracted his disease, was in the Yerba Matté virgin forests. From the same district many peones, or native labourers, come suffering from buba, or *Leishmaniasis americana*. The water used at his station was from a stream running towards the Parana valley. As manager of a plantation, the patient had been able to live in comfort, and feed well. His ordinary diet consisted of fresh meat every day, beans, rice, vegetables, fruit, mandioca, maize, bread. The patient was

a very abstemious man, and, besides, alcoholic liquors are not allowed on the plantations. The patient, recalling his earlier attack twenty years before, and the fact that he had recovered completely within a month and a half, did not agree with my diagnosis that his disease was beriberi, and, besides, no others were known to have suffered from such a disease in that district during the years he had been resident there. Two years previously, however, a case was brought to me from a place a few leagues distant from A. P. C.'s residence, suffering from the dropsical form and with a very bad heart. He unfortunately died shortly after he arrived. I have notes also of a case exactly similar to that of A. P. C., the patient coming from the same district in 1911. He took some six months to get well, and only began to shew marked improvement when he went down to Buenos Aires, a distance of some 1,500 miles. He returned from there completely cured.

I could obtain no history of any epidemic of a similar disease in the district from which these cases had come.

The patient, A. P. C., made an excellent recovery, and when he left was enjoying better health than he had done for many years.

SOME OBSERVATIONS ON AMERICAN LEISHMANIASIS AS A GENERAL INFECTION.

BY

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In a paper by Dr. GEORGE C. LOW on "A Case of Oriental Sore treated by *Antimonium Tartaratum* (Tartar Emetic), locally" *(Journal of Tropical Medicine and Hygiene,* Nov., 1915), the question is asked: Are these sores as strictly local as they have usually been believed to be? Recent observers have thrown doubt upon this, and the American leishmaniasis would seem clearly to shew that a general infection may follow. It is possible that the condition is general from the start, like kala-azar, and that the parasites circulating in the blood settle at different places and produce sores. Blows or abrasions might determine the point of lodgment."

The following histories are those of a dozen cases of American leishmaniasis seen recently in the routine of my practice in Paraguay:—

1. Antonio Recalde. Age 20 years; from the Yerbales or Yerba-Matté plantations; during the last fifteen days two ulcers had appeared, one on the upper left arm behind, and the other slightly below the umbilicus to the left.

2. Maximiono Pedroso. Age 28 years; from the Yerbales; three weeks previously an itching papule appeared in the leg, now it is an ulcer the size of half-a-crown.

3. Romon Rodriguez. Age 30 years; from the Yerbales; five months ago noticed a papule on the right arm; within sixteen days it was a typical buba ulcer. No nasal symptoms had appeared as yet.

4. José Leon. Age 20 years; from the Yerbales; for over a year has had a large buba ulcer two inches above the umbilicus, with nodular

lymphatic cords passing towards the sternum and right nipple. There are no signs of spontaneous healing, and no nasal symptoms.

5. Rufino Lescano. Age 25 years; from the Yerbales; three years ago one ulcer appeared above the right wrist; within three months other three ulcers had appeared above the original one in a line leading towards an enlarged gland at the elbow. The ulcers shew no signs of spontaneous healing, and there are no nasal symptoms.

I quote these cases, because, although the patients were not suffering in their general health in any way, yet the dread they have of the inevitable invasion (as they believe) of the nose and pharynx, makes all such patients seek medical help as soon as they can get leave from their employment.

The following are cases in which the naso-pharyngeal ulceration appeared at varying intervals after the appearance of the initial papule:—

6. José Colman. Age 25 years; from the Yerbales; three months ago two ulcers appeared on the right leg, and one on the left arm, and another to the right of the umbilicus; within two months the ulcers had healed spontaneously, but as they healed nasal symptoms began to appear and have increased.

7. Cecilio Ayala. Age 20 years; from the Yerbales; five months ago a buba papule and ulcer appeared on the right temple; the sore is now cicatrising spontaneously, but the naso-pharyngeal symptoms have appeared and progressed rapidly.

8. Dolores Ayala. Age 35 years; from the Yerbales; six months ago ulcers appeared on the right forearm and left upper arm; these now shew signs of healing, but the nose and upper lip are already badly ulcerated.

9. Julian Medina. Age 27 years; from the Yerbales; had three healed ulcers in leg; these had appeared two years ago, and within a year began healing spontaneously; three months ago nasal symptoms began to appear, and now there is well marked ulceration of the nose and thickening of the soft palate.

10. Emidio Galeano. Age 34 years; from the Yerbales; seven or eight years ago had ulcers on right heel and left hand, which healed spontaneously in four years. In the third year the nose and throat became affected, and now are extensively ulcerated.

The two following cases may illustrate the theory that blows or abrasions may determine the point of lodgment:—

11. Lenon Cristaldo. Age 25 years; from the Yerbales; three years ago was in rather poor health, being very anæmic; in this state he had a small accident, in which the dorsum of the right foot was wounded by a bamboo. The wound instead of healing, developed into a buba ulcer; later, another ulcer appeared over the outer left ankle. Within one year the ulcers had healed spontaneously, but as they healed nasal symptoms appeared, and in the last two years there has been extensive invasion of the naso-pharynx.

12. Dionisio Vega. Age 24 years; from the Yerbales; five years ago his cart upset, and he was wounded on the right inner heel by the zinc roof of the cart. The wound did not heal but degenerated into a buba ulcer, involving the whole of the heel from the inner to the outer aspects. In four years the ulcer healed spontaneously, but about six months before it healed the nose began to be affected, and within a year the whole naso-pharyngo-laryngeal tract has become extensively invaded, the soft palate and uvula thickened and papillated. Over the heel there is a white cicatrix, $3\frac{1}{2}$ inches long by 1 inch broad.

As will be seen from the histories of the cases given, there are great differences in the severity of the infection, the fulminant cases having the secondary ulceration appearing within a few months, the slower cases perhaps only after years.

All the severe cases agree in this, that, coincident with the beginning of spontaneous cicatrisation of the original sore, the secondary naso-bucco-pharyngo-laryngeal symptoms almost inevitably appear.

DIEMENAL IN THE TREATMENT OF MALARIAL FEVER.

BY

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[FROM THE LONDON SCHOOL OF TROPICAL MEDICINE.]

Recently a substance termed "Diemenal," a solution of "colloidal" manganese has been tried in the treatment of malarial fever. In a pamphlet which accompanies the samples of the drug, it is stated that "a number of observations made in the Colonial Hospital at Tamatave, Madagascar, proved that attacks of malarial fever yielded completely to treatment by 'Diemenal,' and the patients were usually discharged at the end of periods varying from eight to fourteen days. Patients so treated suffered no further attacks during their residence in the colony, often for periods as long as two years." It is also stated that if the drug is given during the febrile paroxysm it brings the temperature down to its normal state, and has the great advantage of preventing relapses.

The conclusion is reached that this form of colloidal manganese is a specific for malaria, and that it produces no undesirable effects.

The method of treatment is as follows:—"One injection of 10 cc. daily is given hypodermically for four to six days. After the second or third injection the temperature is normal and remains definitely so. The number of injections is, however, usually carried to six, bearing in mind the importance of immunisation. It is perfectly well tolerated, and the injections are painless."

In the pamphlet no mention is made of blood examinations to determine the effect of the drug on the parasites.

Recently, having procured a sample of the drug, I tried it in the

manner described above in a case of benign tertian malaria (single infection), the treatment being checked by careful blood examinations.

History of Case : O. L., a Dane, had been all over the world, following out his vocation as a sailor. Recently had been in Java, and had visited several of the coast towns. Twelve days after leaving the last of these, on the voyage home, developed fever. Others of the crew got it as well, and though quinine was administered, it was not evidently given in sufficient quantity, as most of the patients were still having attacks of fever on arrival in England. Five of these were admitted into the Royal Albert Dock Hospital, London School of Tropical Medicine, two being benign tertian infections, one a quartan, and two malignant. O. L. was one of the former, and had a temperature of 103° F., benign tertian parasites being found in his blood. He had never suffered from malaria before.

Systems : Nothing of importance. Liver and spleen not enlarged.

Urine : No albumin, blood or sugar.

Fæces : Negative : no protozoa, no ova.

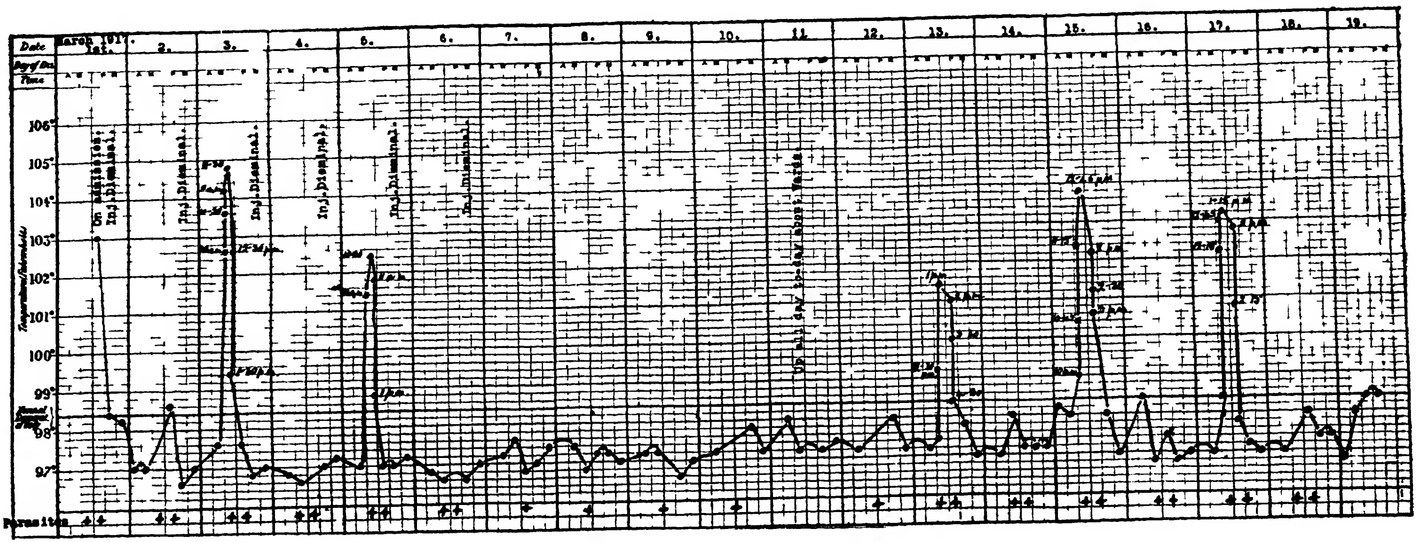
Blood : Benign tertian parasites, gametes and rings, one generation.

Treatment : This was begun at once, on the 1st March, 1917, daily doses of 10 cc. "Diemenal" being administered hypodermically for six days in all. These gave rise to no inconvenience. The patient was at the same time put to bed and kept warm. After the fifth injection the rise of temperature due on the 7th of March did not take place, but the blood examinations shewed that parasites were still present. As keeping such cases rigidly in bed often results in a fall of temperature, irrespective of any drug being given, the patient was ordered up to move about the wards. Rigors began again at once with an increase of the parasites in the blood.

After it was seen that the patient was not cured he was put upon quinine in the usual doses, this stopping further rises of temperature, and quickly causing the parasites to disappear from his blood.

The results of "Diemenal" on this form of malaria cannot, therefore, be said to be encouraging, and much more proof will be required before the statement that it is a specific for malaria can be accepted.

The following chart shews at a glance the temperature, the results of the blood examinations, and the treatment :—


Date
March 1917.
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Normal
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Inj. Diminal.
Up all day to-day about Wards.
Parasites

TRANSACTIONS

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NOTES ON THE PREVALENCE OF INTESTINAL PARASITES IN EAST AFRICA.

BY

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During the last quarter of 1914, a short series of microscopic examinations of the fæces of natives, complaining of intestinal troubles, admitted into or attending at the Native Hospital, Mombasa, were undertaken.

Of 100 examinations, 83 per cent. were positive, 17 per cent. negative. The percentage of the different ova present was as follows :—

Ankylostoma duodenale	46	p.c.
Ascaris lumbricoides	44	,,
Trichocephalus dispar	43	,,
Tænia saginata	29	,,
Schistosoma mansoni	5	,,
Oxyuris vermicularis	2	,,
Strongyloides stercoralis	3	,,

On further investigation during part of 1915 and 1916, a total of 1,500 examinations were made not only of African natives, but of Indians of the Expeditionary Forces and Washihira Arabs of the Arab Rifles.

Total No. of Observations,		1500.	Positive,	51·8	p.c.	Negative, 48·1	p.c.
,,	Indians,	728.	,,	31·7	,,	,, 68·2	,,
,,	Arabs,	83.	,,	62·6	,,	,, 37·3	,,
,,	African,	689.	,,	71·8	,,	,, 28·1	,,

It is necessary to state at the outset that all the subjects examined, Indians, Arabs and Africans, were, as a whole, under conditions inseparable from active service. The percentages of infections may, with certain reservations, therefore be considered to be somewhat higher than they would be under ordinary conditions of life for each of the above races.

No special distinction was drawn in the selection of patients for examination, as was for those shewn in the first table, but cases admitted for all diseases were included.

Considering the Indians of the civilian class following occupations such as those of clerks, railway men, merchants, traders and shop-keepers, it may be said that they rarely suffer from intestinal parasites.

What the figures for these may be it is impossible to say, but one cannot recollect having seen more than three cases during a period extending over two years at Mombasa.

The factors governing this are cleanly habits, thorough cooking of food, protection of the feet by boots, latrines, and a sanitary use of them.

The comparatively high degree of infections among the Indian troops is, without doubt, due to the converse of these conditions holding in the field, and although the different infections were more or less evenly distributed for most areas and seasons of the year, the sick sent in from one area in particular during the latter half of 1915, infected with ankylostomiasis, contributed materially towards raising the total percentage.

The Arab and African, for the class of native admitted, fighting men, followers and carriers, cannot be regarded as abnormally high when compared with work done elsewhere on institutional and village infection under similar sanitary or climatic conditions, or both. African troops, police and domestic servants, suffer far less from helminthic invasion than the villager, whose infection is due to promiscuous defæcation in any patch of scrub he finds convenient. This habit is universal, and probably more than any other factor, accounts for the wide-spread distribution of ankylostomiasis. It is not in any way unlikely that in this manner the fæces of one or two infected individuals may, in a very short time, be the cause of the infection of the inhabitants of a whole village or series of villages.

Some points with reference to the ages of those suffering from ankylostomiasis may be of interest. Children and young adults, unless heavily infected or suffering from some intercurrent disease, do not apply for treatment as frequently as one might suppose. Of this type those who do so do not, as a rule, refer their symptoms to any particular system, but give vague histories of not feeling well or of vertigo, and in the case of police askari usually approach one with the idea of obtaining a change or transfer to stations in their own native districts, the reasons advanced being that the local food, water, salt breezes, or want of them, dependent on the area in which they are at the time of serving, have in some abstruse manner seriously affected their health.

These also frequently develop mental symptoms taking the form of home sickness, melancholia and delusional insanity. In all such instances the fæces should always be examined.

The adult coast native possesses, as a rule, a full knowledge of the disease, and when applying for treatment states that he is suffering from ankylostomiasis, or "safura" as he calls it, and the clinical picture is usually characteristic.

Ankylostomiasis is not uncommonly a terminal infection in the aged.

The first table immediately below gives details of the different causal parasites observed. The other two tables shew the percentages by classes, combined and individual, for the five chief infections:—

	TOTAL.	INDIAN.	AFRICAN.	ARAB.
Total	1500	728	689	63
Positive	778	231	495	52
Negative	722	497	194	31
Ankylostoma duodenale ...	347	110	208	29
Ascaris lumbricoides ...	270	63	195	12
Trichocephalus dispar ...	313	49	235	29
Tænia saginata	168	41	120	7
Schistosoma mansoni ...	29	1	24	4
Oxyuris vermicularis ...	2	1	—	1
Hymenolepis diminuta ...	1	—	—	1
Schistosoma hæmatobium	3	—	2	1
Strongylus subtilis ...	4	3	1	—
Hymenolepis nana ...	2	1	*1	—
Strongyloides stercoralis...	2	—	2	—
Double Infections ...	197	34	146	17
Triple ,, ...	63	3	54	6
Quad. ,, ...	10	—	9	1
Quint. ,, ...	1	—	1	—

* This infection occurred in one of the 19 Cape boys mentioned below.

INDIAN, ARAB AND AFRICAN COMBINED.

Ankylostoma duodenale (Positive)	23·1 p.c.
Ascaris lumbricoides ,, ...	18·0 ,,
Trichocephalus dispar ,, ...	20·8 ,,
Tænia saginata ,, ...	11·2 ,,
Schistosoma mansoni ,, ...	1·9 ,,
Double Infections	13·1 ,,
Triple ,, ...	4·2 ,,
Quad. ,, ...	0·6 ,,
Quint. ,, ...	0·06 ,,

	728 INDIAN.	83 ARAB.	689 AFRICAN.
Ankylostoma duodenale	15·1 p.c.	34·9 p.c.	30·1 p.c.
Ascaris lumbricoides	8·6 ,,	14·4 ,,	28·3 ,,
Trichocephalus dispar	6·7 ,,	34·9 ,,	34·1 ,,
Tænia saginata	5·6 ,,	8·4 ,,	17·4 ,,
Schistosoma mansoni	0·6 ,,	4·8 ,,	3·4 ,,
Double Infections	4·6 ,,	20·4 ,,	21·1 ,,
Triple ,,	0·4 ,,	7·2 ,,	7·8 ,,
Quad. ,,	— ,,	1·2 ,,	1·3 ,,
Quint. ,,	— ,,	— ,,	0·1 ,,

African natives of the following tribes or sub-tribes were examined :—

Wa-Bugu	Wa-Kamba	Wa-Nyika
Wa-Bukedi	Wa-Konde	Wa-Nandi
Wa-Bajun	Wa-Kavirondo	Wa-Zegua
Wa-Barawa	Wa-Kikuyu	Wa-Ziba
Wa-Chaga	Wa-Kisii	Wa-Nyoro
Wa-Dama	Wa-Lendu	Wa-Nyamwezi
Wa-Digo	Wa-Lumbwa	Wa-Nyoma
Wa-Dongoroko	Wa-Yae	Wa-Pokomo
Wa-Embu	Wa-Makua	Wa-Pemba
Wa-Ganda	Wa-Masamba	Wa-Pabai
Wa-Giriama	Wa-Murua	Wa-Sawa
Wa-Swahili	Wa-Meru	Wa-Sagara
Wa-Gazija	Wa-Nubi	Wa-Segeju
Wa-Kakameka	Wa-Nauruma	Wa-Sebei
Wa-Kokea	Wa-Nyassa	Wa-Somali
Wa-Kala		

19 Cape boys are included in the whole total for Africans.

The following table shews percentages of infections among those tribes of which numbers over twenty-five were examined :—

TABLE SHEWING PERCENTAGES OF INFECTIONS AMONG THOSE TRIBES OF WHICH NUMBERS OVER 25 WERE EXAMINED.

The Nubi are here included to shew the high degree of Bilharzial infection among them.

	Wa-Ganda.	Wa-Kavirondo.	Wa-Kamba.	Wa-Kikuyu	Wa-Kisii.	Wa-Nubi.	Wa Nyamwezi	Wa-Somali.	Wa-Swahili.	Total other East Africans.
No. of individuals examined ...	25	166	44	91	61	13	39	21	107	103
Infections—										
A. duodenale ...	24·0	18·0	29·5	36·2	29·5	15·3	48·7	23·8	44·8	32·0
A. lumbricoides	12·0	39·1	22·7	39·5	29·5	15·3	23·0	23·8	18·6	26·2
T. dispar ...	28·0	33·1	23·0	30·7	27·7	33·0	41·0	33 3	47·6	33·0
T. saginata ...	16·0	25·9	18·1	24·1	22·9	7·6	12·8	14·2	8·4	9·1
Schis. mansoni	12·0	3·0	6·8	—	—	30·7	2·5	—	1·8	5·8
Double infection..	24·0	19·8	20·4	18·6	22·9	15·3	28·2	28·5	25·2	20·3
Triple „	4·0	8·4	6·8	12·0	13·1	—	12·8	4·3	8·4	7·7
Quad. „	—	2·4	—	4·3	—	—	2·5	—	—	—
Quint. „	—	—	—	—	—	—	—	—	—	—

It is not, of course, suggested that figures deduced from examinations, varying between 25 to 150 members of any one tribe, examined at a distance from their homes, can be taken as an index of the general and tribal distribution of helminthiasis in East Africa. Nevertheless, curiously enough, in nearly every instance in which the distribution of a disease was previously known, an examination of the last table shews that the natives from those areas return a proportionately high rate of infection to a remarkable degree. For example, bilharzia is well known on the Upper Nile, and a reference to the table immediately shews true by recording a high percentage figure for the tribes therefrom, the Niletic Nubi giving a return of no less than 30·7 per cent.

It has also been recognised for some time past that the natives settled in the Nyika country, along the course of the Kibwezi and Sabaki rivers, are infected with the same disease, and this is borne out by the 6·8 per cent. for *S. mansoni* for the Wa-Kamba.

The Wa-Giriama—the following figures not included in this paper—are also infected, probably very heavily, for of 18 porters sent for physical examination, who appeared to be in good health, a history of previous hæmaturia was obtained from 9, and of 4 of these, who also stated that they could pass blood at the time, and which was done, the terminal-spined ova of *S. hæmatobium* were demonstrated in all.

On the other hand, the high figures for the tapeworm infections, almost all *T. saginata*, are due, I believe, to the personal habits of the natives of certain tribes, *i.e.*, the Wa-Kavirondo, Kikuyu and Kisii, who do not cook their meat sufficiently but eat it practically raw, and their results are therefore correspondingly raised.

Variations in the distribution of the various infections are thus not only dependent upon geographical, climatic, and seasonal conditions, but likewise on the tribal and individual habits of the native.

Parasitic human helminthiasis occupies an important position in its bearing on the question of native labour, more especially with reference to its relationship to the inefficiency and mortality which occurs whenever such labour is concentrated on works, whatever they happen to be.

From the administrative outlook the problem for the present must apparently confine itself to the practical sanitation of townships, stations

and areas, necessarily circumscribed and selected, on which native labourers are employed in large numbers.

Expenditure incurred for such special and temporary reasons, and directed towards the attainment of immediate results, is not only justifiable but economic.

No ultimate and permanent results could be anticipated with reasonable expectation of success were the same principle to be applied to large areas, except at an entirely prohibitive cost, unless there were at the same time a decided advancement in the standard of education and civilisation of the native.

A CASE OF ULCERATING GRANULOMA REFRACTORY TO INTRAVENOUS INJECTIONS OF ANTIMONY, X-RAYS, AND OTHER FORMS OF TREATMENT.

BY

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[FROM THE LONDON SCHOOL OF TROPICAL MEDICINE.]

In the *British Medical Journal*, September 16th, 1916, we published the report of a case of ulcerating granuloma successfully treated by intravenous injections of antimony. In that paper an account of the disease is given, as is also the work of the South Americans on the treatment of the condition by injections of antimony. It is not therefore necessary to enter into these details again here.

Though many successes are recorded, there is not much mention of failures, except in Dr. H. C. DE SOUZA ARAUJO'S thesis on the subject, a review of which appeared in the *British Medical Journal*, July 29th, 1916. Here three relapses are noted, and it is also stated that two old and grave cases died.

Having had the opportunity of treating another case recently, and this time unsuccessfully, we think it is important to report it, as failures in treatment are so often passed over without anything being said about them.

The history of the cases is as follows:—

H., aged 34, male; white man. Once abroad only, having done one tour in Africa, on the Congo. Arrived there 1913. Stayed 31 months, then home.

Previous illnesses.—Malaria severely, cerebral type, no dysentery, no history of syphilis. On the whole enjoyed quite good health.

Present illness.—Five months before leaving Africa had a soft sore on the penis, this appearing eight days after exposure to infection. After this, the glands in the right groin began to swell, but as his right toe was also badly ulcerated at the same time, as the results of jiggers, it was supposed the bubo was due to this. A doctor opened the swelling. Later, the wound instead of healing, broke down, became ulcerative in character, and the ulceration gradually began to spread to the adjacent parts, other foci also starting. Six days after the sore on the penis appeared, he was given a dose of "606," and three weeks after the ulceration began injections of (?) mercury, the doctor evidently concluding the lesion was venereal. No better after these, however. Instead, as just mentioned, the ulceration gradually began to extend and to spread along the groin. After lasting four months, patient was sent home to England, where he arrived on March 12th, 1916. He was admitted into hospital and treated by scraping and escharotics. Again this treatment had no effect, the condition getting worse and spreading down between scrotum and thigh, and then on to the former. At this time he was sent to the London School of Tropical Medicine for treatment, the condition being recognised as a tropical one. He was admitted on the 14th of August, 1916.

Examination.—The patient looked healthy, conjunctiva a good colour, tongue clean, teeth healthy. Heart and lungs normal, no signs of tubercle. Liver enlarged 2 inches below costal margin. Spleen 2½ inches below cm.

Urine.—No albumin, blood or sugar.

Fæces.—Nil, no protozoa, no ova.

Groin.—The whole of the right groin was implicated in an ulcerative process running from the anterior superior spine to the side of the scrotum, the depth of the ulceration being comparatively superficial, with a fibrosed and thickened base. There were, however, signs of attempted healing at the sides and also in parts of the interior. The condition had been much interfered with by the scraping, but even so, it had all the usual characteristics of an ulcerating granuloma, and was diagnosed as such. It did not look like a syphilitic condition, and WASSERMANN'S

reaction was negative on three different occasions. There were no signs of the original sore on the penis, nor were there any ulcerative lesions present on that organ.

It is not altogether clear what this sore was. Possibly it had no connection with the ulcerative lesions which subsequently developed, or what is probable is that it and the ulcerative granuloma infection were acquired at the same exposure, the latter breaking out on the groin after the wound in the bubo had been made. It is common, of course, for ulceration to appear on the groins or pubis with no signs on the penis.

Intravenous injections of antimony tartaratum were begun at once, and the results obtained by these and other forms of treatment are shewn in the following table: –

TABLE OF TREATMENT.

DATE.	DRUG AND DOSE EMPLOYED.	REMARKS.
1916.		
Aug. 14	Antimonium Tartaratum intravenously, gr. 1	Treatment begun to-day.
,, 17	Ditto .. ... gr. 1	Place looks better already.
,, 21	Ditto .. ... gr. 1½	Spreading at one place. Otherwise better, if anything.
,, 24	Ditto ... ... gr. 1½	Looks a little better. Scrapings taken from it to-day and examined.
,, 28	Ditto .. .. gr. 2	Distinctly better, edges less swollen, contracting, floor clean and healthy looking. New places seem arrested. Keeping it dry with dusting powder.
,, 31	Ditto .. ... gr. 2	
Sept. 4	Ditto ... ... gr. 2½	Improvement maintained. Again a little better. Epithelium spreading in now.
,, 7	Ditto ... ... gr. 2½	Improvement continuing.
,, 11	Ditto ... ... gr. 2½	
,, 14	Ditto ... ... gr. 2½	
,, 18	Ditto .. .. gr. 2½	
,, 21	Ditto ... ... gr. 2½	
,, 25	Ditto ... ... gr. 2½	
,, 28	Ditto . ... gr. 2½	
Oct. 2	Ditto ... ... gr. 2½	Great improvement, some parts completely healed.
,, 5	Ditto ... ... gr. 2½	
,, 9	Ditto ... ... gr. 2½	Improvement continuing, but slow.
,, 12	Ditto ... ... gr. 2½	
,, 16	Ditto ... ... gr. 2½	
,, 19	Ditto ... ... gr. 2½	Steadily improving.
,, 23	Ditto ... ... gr. 2½	Further improvement.
,, 26	Ditto ... ... gr. 2½	
Nov. 2	Ditto ... ... gr. 2½	
,, 6	Ditto ... ... gr. 2½	Tendency to break out again. New place over pubis and on scrotum.
,, 13	Ditto ... ... gr. 2½	Not looking well. Frequency of the injections increased, viz., three times a week, Monday, Wednesday and Friday, two grains a time.
,, 15	Ditto .. ... gr. 2	

TABLE OF TREATMENT—*continued.*

DATE.	DRUG AND DOSE EMPLOYED.	REMARKS.
Nov. 17	Antimonium Tartaratum intravenously, gr. 2	
,, 20	Ditto . . ... gr. 2	
,, 22	Ditto .. ... gr. 2	
,, 24	Ditto ... .. gr. 2	
,, 27	Ditto ... gr. 2	
,, 29	Ditto gr. 2	
Dec. 1	Ditto . gr. 2	No better: worse, if anything.
,, 4	Ditto . gr. 2	
,, 6	Ditto ... . gr. 2	Place over pubis increasing in size.
,, 7	First X-Ray exposure ...	
,, 8	Antimonium Tartaratum intravenously, gr. 2	About the same.
,, 11	Ditto . ... gr. 2	
,, 13	Ditto ... .. gr. 2	
,, 15	Ditto . .. gr. 2	
,, 18	Ditto ... gr. 2	Condition much better again, but by no means cured.
,, 19		Antimony injections stopped, and wound now treated as an ordinary septic one.
,, 28	Second X-Ray exposure	Condition a little better again. Improving slowly.
1917.		
Jan. 22		Condition worse; breaking down again. Fresh antimony treatment started.
,, 23	Antimonium Tartaratum intravenously, gr. 2½	
,, 26	Ditto .. ... gr. 2½	
,, 30	Ditto ... . gr. 3	Looking very bad; spreading.
Feb. 2	Ditto . . gr. 3	
,, 6	Ditto ... .. gr. 3	
,, 9	Ditto . gr. 3	No better; worse, if anything.
,, 12	Third X-Ray exposure...	
,, 13	Antimonium Tartaratum intravenously, gr. 3	
,, 16	Ditto . ... gr. 3	Getting worse.
,, 20	Ditto . . ... gr. 3	
,, 23	Ditto . gr. 2	Pains in back, antimony therefore reduced in dose. Antimony stopped after 23rd.
,, 26	Fourth X-Ray exposure	
,, 27	Galyl ·4 gram., Pot. iod. gr. x. t.i.d. ...	Ulcers looking a little better.
Mar. 7	Galyl ·4 gram., Pot. iod. gr. xv. t.i.d. ...	Perhaps a little better, ulcers a little shallower.
,, 12		No better, worse again, anti-specific treatment no effect.
,, 14		Left hospital to-day.

It will thus be seen that the patient had three courses of antimony, viz.: 25 injections August 14th to November 13th, 1916, 56½ grains; 15 injections November 15th to December 18th, 1916, 30 grains; and 10 injections January 23rd to February 23rd, 1917, 28 grains; or a total of 114½ grains in all.

Distinct and marked improvement followed the first injections, but then the condition seemed to become resistant, and instead of healing occurring, further extensions took place, and when the patient left hospital he was as bad, or worse even, than when he entered.

He was strongly urged to remain, but his relatives insisted on removing him, and did so against all advice to the contrary.

A perusal of the table also shews that the patient had four X-ray exposures. The first of these seemed to produce some results, the latter none. In addition, mixed vaccines, made from the organisms growing in the ulcers were administered, and many different varieties of local treatment were also tried. Antimony oxide powder, zinc oxide powder, and calomel, were dusted on, fomentations were employed, the wounds were treated with liq. sodæ chlorinatæ, the edges were scraped, but nothing had the slightest effect. Finally, as a last resort, anti-syphilitic remedies were given a chance, likewise with disappointing results.

As regards diagnosis, the possibility of the condition being something else than ulcerative granuloma was always borne in mind, but clinically it was perfectly typical of this condition, and it was so diagnosed by other members of the staff who were well acquainted with this disease. The depth of the ulcers, their appearance, the fibrosed base, the tendency to heal at places while extending at others, the history of onset, etc., all combined to support this view.

Lupus, rodent ulcer, and malignant disease were debated, but dismissed, as were also blastomycosis and other ulcerative conditions.

Many different organisms were present in the lesions, bodies similar to those described by DONOVAN, CARTER, CLELAND and WISE being easily found.

The impression these gave one was that they were bacterial in nature and certainly not protozoal. They were evidently the same as the *Calymmatobacterium granulomatis* of ARAGAO and VIANNA (1912).

Whether or not these are the real agents of the complaint remains to be proved: their presence, however, was interesting, as they confirmed the opinion that the condition was one of ulcerative granuloma, and not some other disease.

CORRESPONDENCE.

C/o MEDICAL DEPARTMENT, FIJI,

12th March, 1917.

DEAR DR. LOW,

In reference to your letter dated December 8th last [a letter referring to Dr. HARPER'S paper on "Five Hundred and Forty-two Cases of Yaws treated by Kharsivan and Arsenobillon," published in the TRANSACTIONS of February, 1917, Vol. X., No. 4., and asking him if he could be certain of the exclusion of syphilis in his paraframbæsial cases] and my reply dated February 10th, I beg to forward a communication, very kindly written at my request by Dr. LYNCH, together with a short note by myself, which, we venture to hope, you will find of interest.

With many thanks for your stimulating interest in the matter,

I remain, yours very sincerely,

PHILIP HARPER.

YAWS AS A DISTINCT DISEASE IN FIJI.

Practically every Fijian suffers from typical primary and secondary yaws. The disease begins almost always after the sixth month and usually between the second and sixth years. Up till the sixth month the Fijian infant is, as a rule, a veritable prize baby. Infection before the sixth month, or rather before the eruption of teeth, is certainly accidental, but after that time it may be purposive, though it is, I think, usually accidental.

In addition to this disease, practically all Fijians suffer from a train of symptoms, or from isolated symptoms, closely resembling various of the manifestations of tertiary syphilis.

Moreover, aneurism, general paralysis of the insane, and locomotor ataxy are of frequent occurrence among the Fijians, one case perhaps occurring for every thousand of the total population.

The above facts cannot be doubted by any medical man who, as a matter of routine, thoroughly examines his Fijian patients.

As, however, the tertiary and the more remote sequelæ of yaws are not admitted in many quarters outside Fiji, it is as well to examine the only other feasible explanation of the prevalence of tertiary and quaternary lesions, namely, the possibility of the presence of syphilis.

The supposition that syphilis is the cause of these manifestations must necessarily fall to the ground unless we suppose syphilis to be extremely common amongst the Fijians, almost as common, or quite as common, as the writer considers yaws. In fact, the most likely hypothesis for one wishing to prove the syphilitic origin of these tertiary and quaternary diseases amongst the Fijians would be that the disease which the writer considers yaws is really syphilis.

What, then, are the arguments against the general prevalence of syphilis amongst the Fijians at the present time?

1. The absence of manifestations of hereditary syphilis, such as rashes, snuffles, deformities, marasmus, etc., amongst Fijian infants. The writer has attended at least two hundred Fijian confinements, and has never seen any sign of hereditary syphilis in any Fijian infant, either newborn or older.

2. The absence of "Hutchinson's teeth" in Fijian children and adults. The writer has examined many thousands of Fijian mouths and has never seen this manifestation. Periostitis, keratitis, depressed bridge and other signs, which in other races are looked upon as syphilitic in origin, occur in the Fijians only after an attack of primary or secondary yaws.

3. Although the writer has thoroughly examined many thousands of Fijians, both male and female, including the generative organs, he has never seen a case of Hunterian chancre in a Fijian, whether genital or extra-genital, nor has the scar of a previous chancre been seen.

4. Many Europeans live with, or occasionally cohabit with, Fijian women. No such European has been found infected with syphilis. On the other hand, cases of Europeans being infected from Indians are common in Fiji. As long as a European keeps to a Fijian woman he is considered to be quite safe as regards venereal disease.

5. Though in many of the manifestations of yaws, such as periostitis, gummatous ulceration, etc., a single case may be quite easily mistaken for syphilis, yet, when one deals with thousands of cases, it is seen that the march of symptoms and the relative frequency of certain symptoms, such as what we call crab-yaws or the condition of superficial dry dermatitis, are quite different to what obtains in syphilis, as seen amongst the Europeans and Indians in Fiji.

6. The therapeutics of Fijian disease differs, except in the quaternary stages, from the therapeutics of syphilis, the main difference being that the Fijian disease is not nearly so amenable, if it is at all amenable, to mercury. Furthermore, the power of the higher arsenical compounds (such as salvarsan, neo-salvarsan, and, to a less degree, atoxyl and soamin) is far greater in all stages of yaws than it is in syphilis.

PHILIP HARPER, M.R.C.S. Eng.

(Late Sen. Obst. H.P., St. Thomas's Hospital, London),

D.M.O., Ra, Fiji.

YAWS AND SYPHILIS.

In reference to the question of yaws and syphilis and their similarity or identity, it may be considered as practically proved in Fiji that the two diseases are separate, and the reasons given by Dr. Harper, in his paper above, are, to my mind, sufficient proof that the two diseases are separate. Of course, we recognise that both diseases are the results of infection by a spirochæte, but we equally recognise that it is a different organism of the same family that causes each disease, and therefore syphilis and yaws are to be regarded as running parallel to each other, and as both producing symptoms, which, in their secondary and tertiary manifestations, are strangely alike.

It is said, with a good deal of evidence in favour of the assertion, that no Fijian has ever suffered from a Hunterian chancre. I have had one single instance of such a case reported by one of the Medical Officers of the Government Service some years ago. I have also seen a case of congenital syphilis in the child of a Fijian mother by a European father who had syphilis; the mother shewed no signs of disease at all. I have

never seen a Fijian with syphilis in the course of a good many years' experience of Fijian work, both in and out of hospitals in Fiji. I would draw attention to a paper published in the *Annals of Tropical Medicine and Parasitology*, in June, 1915, in which are depicted, under the name gangosa, a series of pictures of what we in Fiji look upon as undoubtedly cases of tertiary yaws. Arguments are advanced in favour of this sequela of tertiary yaws being a separate disease. Up to the present time Medical Officers in Fiji, who see very large numbers of typical cases of the conditions described as gangosa, have no hesitation in considering the condition as an undoubted manifestation of yaws in a late stage. All Fiji cases have suffered from yaws, which is a higher proportion than that quoted by STITT of Kerr, who says that, of 315 cases, 205 shewed yaws scars.

It is an undoubted fact that yaws in Fiji does not respond to mercurial treatment as it is said to respond in some other countries; so much so that Medical Officers in the colony had long since discarded mercurial treatment in favour of iodide of potassium alone, until the comparatively recent introduction of salvarsan and other arsenical intensives, which are so much more rapid in their action; and I think that most Medical Officers who have gained their experience in Fiji will agree with the conclusions in Dr. HARPER'S paper that yaws, as seen in Fiji, is a distinct disease, and is entirely a separate disease from syphilis.

G. W. A. LYNCH, M.B.,

Chief Medical Officer, Fiji.

EXCHANGES.

Anales de la Academia de Ciencias Medicas Fisicas y Naturales de la Habana.
Annali di Medicina Navale e Coloniale.
Annals of Tropical Medicine and Parasitology.
Archives de l'Institut Pasteur Tunis.
Arquivos do Instituto Bacteriologico Camara Pestana.
Bulletin Public Health and Marine Hospital Service of the U.S.
Bulletin of the Tropical Diseases Bureau.
Bulletin de la Société de Pathologie Exotique.
Bulletin de la Société Portuguaise des Sciences Naturelles.
Bulletin de l'Academie Royale de Médecine de Belgique.
het Geneeskundig tydschrift voor Nederlandsch-Indie.
Indian Journal of Medical Research.
Indian Medical Gazette.
Johns Hopkins Hospital Bulletin.
Journal of the Royal Naval Medical Service.
Journal of the Royal Army Medical Corps.
Journal of Tropical Medicine and Hygiene.
Journal of Tropical Veterinary Science.
L'Enseignement Medico Mutuel Internationale.
Memorias do Instituto Oswaldo Cruz.
New Orleans Medical and Surgical Journal.
Philippine Journal of Science.
Revue de Médecine et d'Hygiene Tropicales.
Sanidad y Beneficencia.
Veterinary News.

NOTE.—The above publications lie in the Society's room at 11, Chandos Street, W., and are at the disposal of Fellows.

J. C PHELP AND SON, PRINTERS WALTHAMSTOW

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Proceedings of a Meeting of the Society held on Friday, May 18th, 1917, at 5.30 p.m., at 11, Chandos Street, Cavendish Square, W. 1, Surgeon-General Sir DAVID BRUCE, C.B., A.M.S. *(Vice-President)*, in the Chair.

SPONTANEOUS DISAPPEARANCE OF YELLOW FEVER FROM FAILURE OF THE HUMAN HOST.

BY H. R. CARTER,

Assistant Surgeon-General U.S.A. Public Health Service, Washington, D.C., U.S.A.

Whether the immunity produced by an attack of yellow fever is permanent or temporary is a disputed point among epidemiologists. That it is permanent has been, and is now, the opinion of American epidemiologists and of the older generation of French and English writers who added so much to our knowledge of this disease by their observations in the West Indies and in Africa. There was, however, a very common belief among the laity of localities in which yellow fever was endemic, that leaving such focus of endemicity for a sufficient time would restore the susceptibility to the disease. This belief is alluded to by many writers. It was, I think, universally regarded as erroneous by those *not* living in endemic areas, but given more consideration and quite frequently affirmed by writers who lived in such endemic areas.

It is fair to say that this latter view is the one advocated by most modern writers. A Commission of the Pasteur Institute, working at

Rio, state categorically "that yellow fever is kept up in endemic centres by recurrent attacks among the indigenes," and this view is accepted by other recent French writers as well as by RUBERT BOYCE, SEIDELIN, and the British physicians in Africa.* With this view the writer does not agree, and counts the contrary proven, in so far as a negative is provable. The proof required is that recurrent cases do not occur and hence must be negative. It would take too long to discuss this question, and the evidence, as the writer sees it, is presented in an article published in the *Annals of Tropical Medicine and Parasitology*, Liverpool School of Tropical Medicine, April, 1916, Vol. X., No. 1, "Immunity to Yellow Fever." It is a question of prime importance in the epidemiology of yellow fever, as will be seen later, and of its sanitation as well.†

Assuming then that one attack of yellow fever gives permanent immunity, let us see what must result, and how the result thus deduced agrees with observed facts.

From the known facts of the conveyance of yellow fever, it is obvious that the conditions for the continued existence of yellow fever in a community are three: the parasites, active *Aedes (Stegomyia) calopus*, and susceptible men; all present at the same time, with access to each other.

Parasites exist only in an infected mosquito or in an infected man. They live in the mosquito only during its life, and only a short time—infective to mosquitoes—in man‡

Here, then, are two postulates :—

(1) Since the parasites in the mosquito live only during the life of the host—say, x days—no interval greater than x days may elapse

* In spite of quoting the opinion of the Pasteur Yellow Fever Commission and giving it much weight, CLARAC and SIMOND ("Fièvre Jaune," page 142, *Traité de Pathologie Exotique*, Vol. III.) say: "For our part, if it be permitted to be absolutely affirmative in such a matter, we would say that a grave yellow fever, or even one of moderate gravity, never recurs."

† By the statement that permanent immunity is conferred by one attack is meant that this is generally true, as of small-pox, measles, etc. That permanent immunity is invariably conferred by one attack is not predicated of any disease. "The absolute does not exist in the matter of immunity."

‡ The evidence for the existence of a reservoir host, other than man and the mosquito, to the writer, seems to have no basis in observation. There is, of course, analogy for it. The evidence—of necessity negative—*against* it, in places in which yellow fever has been studied, is considerable. With the data extant the above statement seems justified—in America, at any rate.

between the date when some sick man infective to mosquitoes is bitten by them, and the date when one of the mosquitoes infected by him feeds on a man susceptible to yellow fever, without the death of the parasites, and hence the extinction of yellow fever in that community.

(2) Susceptible people, then, are necessary for the continuance of yellow fever in a community. Such people must not only be present, but must be present under certain conditions of time and place with relation to the stegomyia infected from other people with yellow fever. If in a community there be no susceptible people fulfilling these conditions, yellow fever will disappear.

Now let us consider a community in the tropics in which yellow fever is present, stegomyia abundant and active at all seasons, and with susceptible people. Parasites, of course, are present in those sick of yellow fever, and, since stegomyia are active all the year round, this place will be an *endemic focus* of yellow fever.

Obviously, if, as we hold, one attack of yellow fever produces in general a permanent immunity, such community will have in time no people susceptible to yellow fever left, unless there is an introduction of such people. Yellow fever would then disappear, and, as soon as the infected mosquitoes died off (within our x days), the parasites would disappear, and the community be free from infection. Once free, it would remain free forever, unless the same three factors for conveyance are again brought together. In the natural course of events a new generation would grow up susceptible to yellow fever, susceptible immigrants move in, and stegomyia breed without limit; but unless the *parasite* be again introduced the community would remain free from yellow fever. In such a community—growing naturally—an epidemic would result if parasites are introduced some years after it has been free of infection, the maximum age of the natives then developing yellow fever depending on the length of the interval of freedom. Indeed, yellow fever would doubtless disappear before there were "no people susceptible to yellow fever left," because, under the doctrine of chances, there would be no susceptible people left fulfilling the conditions of time and place mentioned above before there were absolutely none at all—possibly long before.

An immigration of susceptible people, then, is necessary for the continuation of yellow fever in a community, and if this immigration fails, or fails to fulfil certain conditions, yellow fever disappears. This

mechanism the writer has called "the disappearance of yellow fever by the failure of the human host" (page 24, Supplement No. 19, Public Health Reports, "Yellow Fever: Its Epidemiology and Control").

This immigration might be of susceptible people from some other place, or of infants born in the place itself. This is just as true an introduction of susceptible people as the other. As BLAIR says, they "truly are new-comers." If these additions to the susceptible population *conjointly* fulfil the conditions necessary for the continuance of yellow fever, as I have stated them, it will continue; if they do not, it will disappear.

The effect in continuing yellow fever of each class of these additions to the susceptible population—men from outside the community and babies born in it—will depend on many factors, but among others on its amount, increasing for each class as that class increases, but not directly proportionate to those increments.*

Both classes of immigration, then, affect the continuance of yellow fever, and theoretically either one may be sufficient to continue it. Yet the *proportional* effect of the introduction of an adult and the birth of a baby in keeping up the infection is very different. The average baby for some time—say, six months or a year—goes out of the premises in which it is born very little, and hence its chance of contracting yellow fever *from any other place* is small. If it contracts yellow fever from *that* place it will, in all probability, be sick there, and the only bad effect will be to continue for a short time the infection of this place, already infected, and not infect any other one. An adult goes into many parts of the town, and, since stegomyia feed both by day and by night, his chances of contracting yellow fever are manifoldly greater than those of an infant. As he may well contract the disease in one place and be sick in another—the probabilities are good that he will—he will infect the place in which he is sick. Also he may be sick some hours—even a day—before he goes to bed, and thus infect stegomyia in a number of places. If he lives among susceptible people, that is, new-comers, as a stranger is apt to do, he is the more apt to establish a long-continued focus of infection.

* A mathematician would say that the effect of each class varies as a direct function of the number added by that class, but that the functions for the two classes were not the same, and that their joint effect varies as some other functions of these numbers, but always direct functions, so that both increase or diminish together.

On all accounts, then, the effect of an adult immigrant in continuing yellow fever in a community is greater than that of a baby, and a larger number of babies would be required to keep up yellow fever than of susceptible adults.

Besides the influence of infancy for our native-born baby we must consider his environment. As yellow fever is permanently endemic in the community, all adults native-born or of reasonably long residence are immune to it, and the only yellow fever among adults is among newcomers. An infant born to an immune household is, then, extremely little exposed to yellow fever. He is protected from it by what GUITÉRAS characterises as "the shield of immunity" of his environment. True enough, his brother—five or ten years old and not yet immune—may contract yellow fever elsewhere, be sick at home and infect the mosquitoes there, and the infant contract it. Yet his chance of escape—already good on account of infancy—is much increased by this "shield of immunity" which surrounds him. His chance of contracting it as he grows older is also good—a certainty indeed if yellow fever continues in the community.

Now in such towns the majority—the very great majority—of infants are born in immune households, and it is obvious that to supply the susceptible people necessary to keep up yellow fever it would require a very much larger number of infants to be born per annum than it would of susceptible adults to come in. Adult immigrants, then, should be of supreme importance in keeping up yellow fever, far more so in proportion to their numbers than the native-born infants. In general, both acting conjointly are factors in so doing.

Now what facts do we find consistent with the above theoretical deductions? Yellow fever has disappeared from a number of the towns of the tropics in which no sanitary work has been done and in which stegomyia is still abundant, or before sanitary work was done and while the mosquito was abundant. Corinto was unquestionably infected with yellow fever in 1905, and we received passengers in Panama with yellow fever contracted in that port. We had an inspector in Corinto up to 1911. No sanitary work was ever done there and stegomyia were abundant all the time, although no evidence of yellow fever was seen. In 1912 we landed some 5,000 marines in Corinto, who made the place a base of operations. No yellow fever appeared among them. Evidently the town

was not infected. Yellow fever disappeared from Bocas del Toro *before* any sanitary work was done there and while stegomyia were abundant. The same is true of San Juan, in Porto Rico, where stegomyia are abundant—the disease disappeared *before* the American occupation. After the focus of Havana was removed the same thing happened in many Cuban towns: Matanzas, Villa Clara and Puerto-Principe—which had quite an epidemic in 1899—and even Cienfuegos Carthagena, Baranquilla and Santa Martà have all been infected with yellow fever, and not many years ago. The first was long notorious as an endemic focus. It was the port through which the susceptible travel between Spain and the Colombian plateau, and later was an entrepot for people from the plateau going to Europe and for troops to Panama. The focus at Panama too was active until 1906. A careful examination last summer shewed this section—the Magdalena Delta—to be free from yellow fever, certainly since 1911. Stegomyia were numerous and active in Carthagena; present in fair numbers, but fewer, in Baranquilla (Colombia), which registered a severe epidemic—the first one—in 1911-12; or in Cucuto or in Maricaibo—both of evil fame formerly—but the same search was not made as at the towns of Magdalena Delta, so that this will not be asserted. It is certainly absent at La Guira, yet stegomyia abound. It is not fair to count Caracas, because, although stegomyia are moderately abundant, yet anti-mosquito work was done during the epidemic of 1911-14. Yellow fever was epidemic in Bahia, introduced from New Orleans in 1842 to 1851, "over 100,000 persons being attacked"—an estimate presumably. It lasted until 1862 and disappeared completely, to be reintroduced from Havana in 1869.*

It scarcely seemed necessary to mention the disappearance of yellow fever from the Windward Islands—St. Thomas, Dominica, St. Lucia, St. Vincent and Barbados—or from Georgetown and Demarara, at all of which it has been epidemic. This happened before 1900. At none of these places was its disappearance due to sanitary measures, unless the cessation of European immigration may be so accounted. This disappearance of yellow fever is certainly a not uncommon phenomenon in tropical countries. Indeed, for yellow fever to disappear, to disappear completely,

*This disease is reported to have been introduced once before and to have disappeared, but the nature of the disease of which this is predicated is questioned by some. I do not, from the description given in contemporary reports.

from isolated communities of small or moderate size *is the rule*, and that without sanitary work or diminution of stegomyia.

The writer would explain this by the lack of susceptible people sufficient to continue the life of the parasite—the "failure of the human host"—of the previous paragraphs. The *fact* asserted, that yellow fever does disappear from towns and districts in the tropics without sanitary work and with mosquitoes abundant, is true. This cannot be questioned; there are too many instances. The explanation given above is believed to be true also, because (1) it is logical and accords with what we know, and (2) none other is apparent. Yet it is but a deduction from a fact and not the fact itself. It is submitted then to tropical sanitarians for criticism and investigation.

It is obvious that this explanation depends absolutely on the doctrine that an attack of yellow fever confers immunity against another attack. In proportion as that immunity is permanent and general, the chance of the exhaustion of the susceptible human material by a definite number of cases of fever, that is at a definite time, to the point of causing the disappearance of disease is greater than if the immunity be of short duration and uncertain. If this immunity be not permanent, but yet endures for some time, the disappearance of yellow fever by the mechanism I have outlined can still occur. This is evident. It is also evident that the men in our community who lose their immunity through lapse of time (or any other cause) keep up the supply of susceptible material just as immigrants would. In proportion as attacks recur frequently and at short intervals, so will the chance of failure of the human host, to the point of causing disappearance of the disease, diminish. If "frequently recurring attacks of the indigenes" are the rule, and these recurrences are indefinite, they might very well continue the fever in a very mild or "larval" form indefinitely, independently of immigration or new births. The *possibility* of disappearance by the mechanism we have given is then not dependent upon the permanence of the immunity given by one attack, but the *chance* of its occurring in any place at any definite time is directly dependent on it, and reaches its maximum when, as we believe, one attack gives permanent immunity. The frequency with which yellow fever has disappeared when immigration was lacking is evidence against recurrent attacks.

It can be asserted that the towns which did not free themselves have

been large towns, and with considerable susceptible adult immigration—as Guayaquil, Havana, Rio, Panama, etc. The same is true, to an extent, of towns and places which were in such relation with other towns infected with yellow fever that there is opportunity for frequent introduction of the parasites; hence the use of the word "isolated" in previous paragraph.

The travel-relations between a number of small towns can well be such as to make the region of the combined communities a permanent focus of yellow fever, even if no one of them could continue as such by itself. The conditions must be such as to allow of the mutual interchange of infections (parasites) between the towns and yet not such that the susceptible people necessary to continue the disease will be exhausted in all of the towns at any one time. In such an area, the rapidity of spread of the fever—and hence the exhaustion of the susceptible material—would be much slower than if all were in one town. There might be intervals long enough between infections in a community for a considerable number of natives susceptible to yellow fever to accumulate. This would give rise to a series of small epidemics among the children, of age dependent on the length of the intervals of freedom from yellow fever of that community. This may be the explanation of the persistence of yellow fever in Yucatan and Campeche, and in Africa, and appears to have been the case with the littoral of the Caribbean Sea before Havana became a permanent focus—in 1761—no one port being *continuously* infected, but some port in this area being infected at all times, the area *as a whole* being a permanent endemic focus of yellow fever. Such a conception seems to the writer to best explain the history of yellow fever on the Spanish Main and West Indies during this period. There is nothing unusual in this conception. It is analogous to the continuous existence of measles in a section of country comprising a number of towns, no *one* of which is always infected with it. The elimination of the insect host in several, or even one, of these communities, to such a degree as to make them not infectable with yellow fever, may so break the chain of transfers of infection as to allow of all the rest of this communicating area freeing itself permanently by the failure of the human host. Anti-mosquito work then at even a few of such places may give results good out of proportion to its extent.

The effect of a permanent focus, as Havana became in 1761, was not

to *prevent* other communities freeing themselves from yellow fever by the mechanism we have outlined, but to *re-infect* them as soon as a sufficient number of susceptible people—by birth or immigration—had accumulated to allow of propagation of the fever. These smaller places, then, shew a series of small epidemics, with periods of freedom from fever between them. The towns on the Guayaquil and Quito Railroad, and on the west coast of South America, in communication with Guayaquil, illustrate this beautifully. There was an epidemic in Buenaventura in 1903 and 1904; freedom for some years; then in Tumaco and Esmaraldas in 1914; and again in Buenaventura in 1915 and 1916. Only "children and people who had moved in during the last eight or ten years" were attacked.

It is this recurrence of fever which has given rise to the belief in "larval" yellow fever held by many very eminent men. Unquestionably yellow fever may, and at times does, exist unrecognised among the native children of a community; shewing itself only—or rather being recognised only—when it attacks some stranger. Here we have true recrudescence whenever an influx of strangers occur.

This view is too well known to require elaboration. It is true, and the writer will not pretend to predicate how long such a condition may last; nor deny that, under some conditions, it may last indefinitely, and by this means alone keep the place or area of communicating places a permanent focus of yellow fever. What he does deny is that this is the rule. The instances given prove that the spontaneous disappearance of yellow fever is not rare. An analysis of them, if there were space, would shew that in the absence of adult immigration and of inter-travel—if one may coin a word—among them, this is to be expected to occur in a large proportion of towns, and that after this even when there are influxes of strangers, outbreaks in such towns do not occur, unless they are in communication with some infected focus. These outbreaks then are re-infections, and not recrudescences of larval yellow fever.

For a town, therefore, which has freed itself from yellow fever *by the failure of the human host* to remain *permanently* free from yellow fever, *isolation* from infected places is necessary. When yellow fever has been eliminated *by the control of the insect host*, this isolation is not necessary *as long as this control continues to be efficient*, because, in the absence of the insect host, yellow fever is not communicable, and such parasites

as are brought in by infected men or infected mosquitoes would, at the most, establish a very temporary focus of infection. If the control were complete, infected men would transmit no parasites. From infected mosquitoes men would indeed be infected, but from them there would be no transmission to other mosquitoes, and hence no secondary human cases, and with the death of the mosquitoes introduced the conveyance of the disease would be over. It is to be noted, however, that the reduction of stegomyia sufficient to eliminate yellow fever from a town in the tropics would nearly always be less than may be required some years later to prevent its spread, because there will then be a larger proportion of susceptible people than at first. Hence the mosquito control must be more intensive.*

What has been said explains the great diminution of yellow fever which is now apparent in the Americas :—

(1) The extinction of the great permanent foci of Havana, Vera Cruz, Panama and Rio. With this a number of less important places were freed from yellow fever by sanitary work in control of the insect host: Para, Manaos, Iquitos, probably Pernambuco, and possibly Bahia and Caracas. This enabled such ports, on the Caribbean especially, as could spontaneously free themselves of yellow fever to remain free—being no longer exposed to infection, or rather much less so exposed.

(2) The substitution of steam for sailing vessels had already enormously lessened the number of infections (parasites) carried between ports, thus lessening the re-infection of such ports as had cleared themselves of yellow fever. Sailing vessels frequently carried stegomyia as well as infected men, while iron steam vessels very rarely carried the former, and hence, spite of the shortened voyage, were much less efficient in the transport of parasites.†

(3) The European war both restricted foreign immigration and, by the commercial depression it caused, greatly lessened the movement of

* It is worth contrasting these two methods for the elimination of yellow fever. That by the control of the insect host is unquestionably the method of election for the sanitarian, while the method by which it is eliminated *in nature* is by failure (control) of the human host. (Supplement No. 19, U.S. Public Health Reports, pages 10 to 12, "Yellow Fever," Two Lectures, etc., H. R. CARTER, pages 10 to 14).

† *New York Medical Record*, March 22nd, 1902, "Are Vessels Infected with Yellow Fever? Personal Observations." H. R. CARTER. *New York Medical Record*, May 14th, 1914, "Some Characteristics of *Stegomyia fasciata* which affect its Conveyance of Yellow Fever." H. R. CARTER.

all people between different towns and from the plateaus to the coast towns—thus both lessened the number of susceptible people in infected places, and by lessening travel limited the chance of spread of infection to places which had cleared themselves of infection. Where the parasite is absent the fever will not recrudesce with a return of prosperity and influx of susceptible people. These have returned to Maracaibo and to some other towns with no outbreak of fever.

It is facts like the above which make us so hopeful that a reasonably well-organised effort against yellow fever will result in its elimination from the earth—its complete elimination never to return. In many places where yellow fever now exists, it will need but a minimal amount of sanitary work to turn the scale against it and eliminate it, and the freeing of one place from yellow fever so frequently prevents infection of another—an endless chain for good. And there has never, since its history began, been a time so favourable for making this effort as the present. An attack on a weakened, retreating enemy pressed home, should not admit of rally, but end in his complete destruction. It is upon these facts that the recommendations of the Yellow Fever Commission of the International Health Commission have been based. The purpose of these recommendations is this complete, permanent elimination of yellow fever from the globe. This fate has befallen some of the higher forms of animal life, even in historical times; but this will be the first time in which an attempt for this purpose is made against a micro-organism pathogenic to man. Its accomplishment will (I do not say "would") mark an epoch in sanitation.

Discussion.

Sir James Kingston Fowler: I feel great diffidence in speaking before the Society of Tropical Medicine. You gentlemen know about these diseases actually. I only hear them spoken of and read about them. I had hoped when I, rather carelessly, read the notice of the meeting that I should have the pleasure of meeting Dr. Carter here. I have long desired to meet him. His observation on the extrinsic period of incubation in yellow fever is one of the most remakable observations I know of, and he is a very great authority on that disease. The Yellow Fever Commission, West Africa, had to consider the point

which was raised by Sir RUBERT BOYCE, and is referred to in this paper, viz.: How is yellow fever continued in the intervals between epidemics? BOYCE believed that it was continued by a constant succession of attacks in natives in childhood, again in youth, and so on. That view was not accepted by the Commission. I am glad to find that the view we adopted is supported by such an authority as Dr. CARTER. Sir RUBERT BOYCE likened yellow fever to malaria; he spoke of it as a sister disease to malaria. Malaria is always present in West Africa, and it is in nearly every person. We could not find any evidence that yellow fever was present in that way. We concluded that there were endemic foci and endemic areas in which the disease was kept alive, but that it was not of universal prevalence. We went so far as to name a very considerable number of places which we thought were foci of yellow fever. By a focus I understand a small spot from which the disease is never absent, and by an area something like the author mentions here, *i.e.*, a number of adjacent places from one to the other of which the disease passes. We concluded that the belief in the immunity of the native to yellow fever is founded on an error of observation: he is not immune to it, he simply has the disease in a mild form. This is the rule, though it is not invariable. Persons who live in this country have measles in a mild form, the reason, being, I take it, that measles has been present in this country from time immemorial, but it is not present in every village in the country at the present moment, although it has been there at some time or another. The resident in this country is not immune to measles. I suppose immunity to yellow fever is analogous to the immunity to typhoid which is ensured by the use of an antityphoid or triple vaccine, and we know that that immunity dies out. Some observations were made in a hospital in West Africa on willing natives who knew the disease well and were not afraid of it. Inoculations were made, and every one of the natives inoculated took the disease. They said: "We know this well: white man die; small fever for black man." These natives, as a result of the inoculation, developed a mild attack of yellow fever, therefore they were not immune.

I would draw the attention of the meeting to another point. I have lately had to consider an outbreak of fever in Barbados, which, in this paper, is stated to be free from yellow fever. I have not time to go into that, but there has been an epidemic there lately, which has been investi-

gated by Dr. GUITÉRAS, who wrote a letter to some resident doctors, in which he gave his reasons for concluding that the epidemic was not one of yellow fever. It is the only instance I know of in which an outside observer has held the opinion that an epidemic was not yellow fever, whereas a considerable number of the medical men on the spot have believed it to be so. It is suggested in the letter that the disease may have been "Japanese jaundice," that is spirochetosis icterohæmorrhagica (Weil's disease). But there is no statement as to observations on the urine, or on the blood, or of injections into guinea-pigs. When the Fourth Report of the Commission was written I had not seen a case of spirochetosis icterohæmorrhagica, but I have since seen many in France. The first thing which strikes persons gaining clinical experience of that disease is the frequent occurrence of hæmorrhagic herpes, yet it is not mentioned in the letter; and in all my reading I have not met with a single mention of it in association with yellow fever. If yellow fever and Weil's disease were the same it is a million-to-one chance that that fact would have been observed, but it has not been observed.

Another clinical point is that in Weil's disease the voice of the patient may dwindle to nothing—a most remarkable feature; but neither have I seen that mentioned anywhere. This weakness of the voice is not due to extreme weakness in the individual. It would, I believe, be possible to place in parallel columns the symptoms of yellow fever and those of a very severe case of Weil's disease, and it would appear that the two diseases must be the same. Yet those who have had clinical experience of both diseases—I have not—would, I believe, be able to differentiate them immediately. There is no mention of the condition of the voice in the disease observed in Barbados which is referred to as Japanese jaundice, nor is there any mention of hæmorrhagic herpes. Therefore I conclude that the disease was not spirochetosis icterohæmorrhagica. I do not assert that it was yellow fever, but it may have been.

Professor W. J. SIMPSON: I do not care about occupying the time of those present, it being so late. Most of my observations corroborate what has been stated. I was always an opponent of the theory that was brought forward at this Society by Sir RUBERT BOYCE; and the more experience one had of the epidemics which occurred in West Africa, the more one was convinced that the attacks were kept up—mainly, at all

events—by travel and communication from place to place. Towns were free of the disease for a very long time, and then suddenly a case occurred and there was an outbreak. I shall not labour that any further.

I think that when Surgeon-General CARTER speaks about the disappearance of yellow fever he is correct in a way. Many of these towns had it for a number of years and then it gradually died out; there was no communication with infected centres. Still, if he claims it as a thing which is likely to occur in practice, I fear it is a doctrine which may be misunderstood by the laity. I remember very well, in regard to India, that when plague broke out there it was said by a large number of people: "It is all right: the first year is bad, the second is not; it will have died out." There were ten million people in their graves as a disproof of that. I think the case is the same with yellow fever: it may easily recur in a large town. I mention that as a warning in regard to these matters; and the curious thing is that Surgeon-General CARTER, when he comes to deal with yellow fever, employs sanitary measures. I take it that isolation of the sick, the prevention of importation, and the supervision of those who come from infected areas, as well as the anti-mosquito measures, are all sanitary procedures Sanitation does not merely mean looking after a certain number of drains or some scavenging; it is on a much broader basis than that, and I have no doubt that as a result of these measures being carried out, in West Africa also, a great change will take place. The statement that the disease will die out is against epidemiological ideas at the present time. The best illustration of that is that thirty years ago everyone said, "Plague is as dead as the dodo." Yet it is spreading all over the world now, and I have no doubt its manifestations will become much worse even than they are now. I do not think any disease dies out of itself. A certain latency may exist for years, but this is often followed by recurrences on a large scale.

Dr. G. C. LOW: I have listened with great interest to Sir JAMES KINGSTON FOWLER'S remarks with regard to the recent epidemic of yellow fever in Barbados. I did not quite understand whether it was the Barbados physicians or Dr. GUITÉRAS who said it might be Weil's disease or Japanese jaundice.

Sir J. K. FOWLER: GUITÉRAS leans to the idea that it was not

yellow fever. He suggests rather that it was Weil's disease, though he seems to have made no investigations to prove it.

Dr. G. C. Low: My interest in yellow fever dates back to 1901, when I was in the West Indies, and I suppose I must congratulate myself upon having had the opportunity of seeing an epidemic of this disease, and at the same time having had the opportunity of studying the condition in detail. Comparatively few of our present tropical workers have had such a chance, and still fewer will in the future, so rare is the disease becoming as the result of improved sanitation.

When I was in Barbados (1901) I was informed of an epidemic that had occurred in the island ten years or so before. Negroes suffered from and in certain instances died of it, black vomit being a feature of some of the cases.

In Dr. CARTER'S very interesting paper I notice that he mentions the West Indian Islands, and states that in none of these cases was the disappearance of yellow fever due to sanitary measures, this happening before 1900. My epidemic, if I may term it so, occurred in St. Lucia in 1901, and I think its control and rapid disappearance may with certainty be claimed to be due to the sanitary measures that were adopted by the Army doctors and myself in dealing with it. This small epidemic has escaped the notice of most people, Dr. CARTER probably included, owing to the fact that it has never been published in the medical journals, though there must be records of it in the archives of the War Office. It is specially interesting, as the dicta laid down by the American Commission in Havana, which we had just received, were followed out in detail, and what might have been a serious outbreak was limited instead to comparatively small proportions. Major WILL and Major BENT, R.A.M.C., have both passed away since those days, and only Captain MCGRIGOR and myself now remain of the workers who took chief part in the work. Briefly the history of this epidemic is as follows. I was in Barbados in the beginning of December, 1901, when it was officially notified that yellow fever had broken out in St. Lucia. Sir FREDERICK HODGSON asked me if I would go over and lend a hand, so, after having seen the General and his staff, I sailed for St. Lucia the next day. What had happened was that among the troops stationed at the Morne, a hill 800 feet or so above the town, several deaths had occurred during the months

of August, September, October and November, some of the latter presenting suspicious signs of yellow fever. Malaria, it is true, was exceedingly prevalent, but no similar ratio of deaths had taken place in previous years when that disease was just as abundant. In December four deaths occurred in one day, the symptoms of those being perfectly typical of yellow fever, vomiting of black material, etc., so the disease was officially notified and the island placed in quarantine. I arrived just after these cases had been buried. For a time there was a lull, no further fresh cases developing, though there were several still in a convalescent condition. The usual difficulty of excluding malaria in many of the cases cropped up, quinine having been given to all immediately on signs of fever appearing. I got an order passed that no one should have this drug until blood examinations had been made, and one was then in a better position to determine whether the case was one of malaria or yellow fever. After a time another case appeared, this time at La Toc, a considerable distance from the quarantined area. This patient had been in St. Lucia for a considerable time and had had malaria, and, as a matter of fact, had a few scanty malignant rings in his blood the morning he took ill. Death occurred suddenly on the following morning. At the autopsy the liver was of a typical boxwood colour, and there was black grumous liquid in his stomach. There is no doubt this was a case of yellow fever attacking a person already the subject of malarial fever. Corroboration of this was soon forthcoming; a few days later two other officers in the next house, twenty yards or so away, sickened and went down with fever. One recovered the other died of typical yellow fever. He became progressively yellower, the urine was loaded with albumen, petechial hæmorrhages appeared, and, though actual black vomiting did not occur during life, the stomach post-mortem was full of that material. There was also a well-marked post-mortem rise of temperature. We knew of Weil's disease then and read the subject up, Major WILL and myself often discussing this and other diseases that might possibly be mistaken for yellow fever. Hæmmorrhagic herpes never occurred in any of our cases, and there is no doubt the disease was real yellow fever.

The sanitary measures we adopted for controlling the epidemic were based upon the American work in Havana. The troops were moved to another area and no one was left inside the infective zone, with the exception of the doctors and two or three other white people, for carrying

on the drainage work and fumigation. We took the houses where cases had occurred first, and carefully fumigated them with sulphur or pyrethrum, and then dealt with the others until all had been done. The breeding grounds of the stegomyia were also dealt with, and drainage and other sanitary measures were carried out. Amongst the troops moved two suspicious cases occurred, but from the moment they took ill they were kept under mosquito nets, and no other cases developed.

Now how did this outbreak originate? On arrival in the island I worked through all the official papers, and finally, I think, got to the root of the matter. I found out that a white sergeant of police, whose duty it was to inspect ships at St. Lucia when they came in from the Brazilian ports, had developed fever and died of it some time during the later part of the summer of 1901. The Colonial Surgeon strictly isolated him during the last days of his illness, which shews that he suspected yellow fever and not malaria. I found out that this man on the second day of his illness had spent the greater part of that day in the sergeants' mess at the Morne, where the yellow fever epidemic eventually broke out. From that time cases began to die; not many, it is true, but the death-rates gradually mounted until, as I have already stated, four cases died in one day (December, 1901) amongst the troops, all with black vomit.

Post-mortems were not held on the earlier cases, and their death certificates were signed as malaria.

The original case was evidently infected by an infective mosquito from one of the ships, which had come from one of the infective Brazilian ports. It was owing to the prompt action of Major WILL in moving the troops quickly, when he determined that yellow fever was present, that saved a big epidemic. The people in Castries, the main town, remained free, no cases developing amongst them.

Sir J. K. FOWLER: What Dr. Low has just said is extremely interesting to me. To conclude, as is done in this paper, that Barbados has been free from yellow fever for a long time is dangerous.

Dr. G. C. LOW: I saw the so-called yellow fever houses in Barbados and I know the places. I think there is a photograph of one of them in Sir RUBERT BOYCE'S book. In a case, described to me, that had died in

one of them, no post-mortem was made, and the diagnosis, therefore, is not certain. The house was in the country, far away from the town and shipping, and it is extremely difficult to see how a fresh case could arise *de novo* in such a position.

Sir R. HAVELOCK CHARLES: I wish to thank Dr. Low for having brought this subject up. With regard to the outbreak at Barbados, it resembles, in some ways, an outbreak which occurred in Swansea many years ago. The manner in which the disease spread and the errors made, cause one to be strongly of opinion that if such can occur in England, all the more reason to adopt the most stringent precautions in a country such as India, where the conditions are more favourable and sanitation not so advanced as in England.

What I am most struck by in this paper is its great optimism. Some years ago, in connection with the approaching opening of the Panama Canal, fear was struck into the hearts of those responsible for public health in Asia, by the statement that when the canal would open yellow fever would arrive in Asia. The writer of this paper says it is very rare for yellow fever to travel in iron steamers, but we know that is the way in which it makes its progress up and down the coasts of America. If it can do that, it is still possible for it to start from the Mexican coast and arrive in the Philippines and outlying points of Asia, where there would be found the three factors for the disease.

A practical point is, supposing you are called upon to advise in the Philippine Islands with regard to this disease. A ship arrives from an infected fort: will you allow a passenger on that vessel to land at once, because there has been according to the statement of the master of the ship (no doctor being carried) apparently no cases on board that ship? Or, will you quarantine that ship for so many days? I ask for an expression of opinion from this meeting on that subject, because it is a practical one. There is much theory in the paper, but a straight question brings us to the bed-rock of practice. The Government of India is interested in this matter on account of the great commercial agencies involved.

Having the terrible lesson of the introduction of plague from Hong Kong to Bombay before one, no one responsible for the lives and welfare of the peoples of India wishes to take any risk, no matter how optimistic

may be the voices of some, and no matter how charmingly the upholders of unrestricted commerce may pipe their song—"There is no danger." "The disease is dying away." "Look at what has been done at wiping out this scourge." "India surely can cope with it by proper sanitation if the disease does arrive." "Why adopt any other methods than are found efficient here?"

It must not be forgotten that till you have sanitary science fully applied, you can not rely on it for protection. Quarantine must be used for a time. It is not the ideal method, but till the ideal has been made practical by expenditure of money, and the full working of a department of health co-ordinating full and sound hygiene at the various ports, protection against yellow fever in India must, as I said, be sought in quarantine.

Dr. DANIEL ANDERSON: When I was on the Pacific Coast in 1912, American quarantine doctors came on board and saw that there were no cases of yellow fever amongst passengers or crew; they took great care to see to that, and when we approached some of the towns where yellow fever had been prevalent, we were not allowed to land. At Tokopelia, on the northern coast of Chili, there was a great epidemic of yellow fever in 1911, and Professor PORTER of Santiago told me he felt certain that fever had been brought from Guayaquil by the steamers, and having been sent there to stop it he took measures to disinfect the town. He put the inhabitants in tents on the mountain-side, and disinfected every house, and offered the people money to bring the stegomyia to him. He said that towards the end of his stay there—three months—there was not a single stegomyia to be got, even though he offered two (Chilian) dollars apiece for them. Thus, by disinfection and by the destruction of the stegomyia, the epidemic was stopped. There is no doubt that your *iron* steamers can convey the stegomyia long distances. I constantly found mosquitoes in my cabin, both along the Pacific Coast and in the Caribbean Sea. They had carried some on board whilst we were in harbour.

Sir R. HAVELOCK CHARLES: A practical point of importance to this Society is, that, at the International Sanitary Conference at Paris, the non-necessity of quarantine with regard to yellow fever was stated. That meant that they would allow a vessel to arrive from an infected

port, even with the stegomyia on board, and allow the passengers to land among a susceptible people.

Lt.-Col. S. P. JAMES: I am an official of the Government of India, and I have written a report on the possibility of the spread of yellow fever to India after the Panama Canal is open. I agree with Sir HAVELOCK CHARLES about the extreme importance of deciding what should be done with ships and people arriving at and departing from certain ports, especially in the Philippines, and at Shanghai, Hong Kong and Singapore. One recommendation I made in my report was that the Home Government should place specially qualified officers at the more important ports on the route from the Mexican coast to India. That has not yet been done, but I hope that it will be done in the future.

As regards Dr. CARTER'S admirable paper, my own feeling is that I should hesitate to base a preventive policy on the theory by which he explains the disappearance of yellow fever from certain areas. His theory implies that the yellow fever germ cannot live in the blood of an "immune," and he remarks that Nature's method of eliminating yellow fever is by this destruction of the parasites in the human immune host. But in Nature it is not usual, I think, for parasites to select hosts which cause their own destruction; and perhaps it is more probable that immunity from yellow fever may be no more than immunity from symptoms. At any rate I do not know of any experiments which prove that in the areas from which yellow fever is said to have disappeared there are no "healthy carriers" of the germ.

Professor SIMPSON: I am very much of the same opinion on this matter as Sir HAVELOCK CHARLES. I think there is too much attention paid to the commercial aspect in regard to these diseases. I think the suggestion of Colonel JAMES, which has been carried out by the American Government, in placing physicians at suspicious ports, should be carried out by placing a British India physician at the different ports, and that they should be given power to put certain people into quarantine if they consider it necessary. Certainly all the ships from those ports should be fumigated and thoroughly disinfected. I think it is necessary to carry out Colonel JAMES' suggestion for the safety of the East.

Sir J. K. FOWLER: Colonel GORGAS gave evidence before the Yellow Fever Commission, and I asked him that question [the danger of the introduction of yellow fever into Asia.] He said he thought that so long as the supervision and the precautions now adopted at the Panama Canal were continued, there would not be any danger. He implied, by his answer, that if there were any relaxation of those precautions, there would be danger. There cannot be any doubt that the appointment of British health officers, whose duty would be to watch those ports, is a most desirable measure.

Lt.-Colonel JAMES: In 1912, when I visited the Canal, I understood that much of the vigorous antimosquito work would shortly be discontinued, and after that the canal was completed and opened, the antimosquito measures might not be so good as they had been during the course of construction. That, of course, does not affect the policy of placing American health officers at the different ports: they have their medical officers at Panama, Colon, Hong Kong, and so on; and these officers have power to exercise effective health control of ships from suspicious ports.

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Invented by MRS. W. J. SIMPSON.

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THE DISTRIBUTION AMONG FOODSTUFFS (ESPECIALLY THOSE SUITABLE FOR THE RATIONING OF ARMIES) OF THE SUBSTANCES REQUIRED FOR THE PREVENTION OF *(A)* BERIBERI AND *(B)* SCURVY.

BY HARRIETTE CHICK AND MARGARET HUME,

From the Lister Institute of Preventive Medicine.

NOTE.—This paper has been unavoidably held back. The authors have taken the opportunity to make some additions, *e.g.*, references to the Mesopotamia Commission Report and an Appendix.

CONTENTS.

I. INTRODUCTION.

Sufficient evidence is now available to permit beriberi and scurvy to be classed among the "deficiency" diseases due to a defective diet without further comment. The view that scurvy is caused by the absence of fresh food, especially vegetables, was established in popular opinion more than 150 years ago,* and is maintained to-day, in spite of various attempts, some of which are quite recent (*e.g.*, JACKSON and HARLEY, 1900, and COPLANS, 1904), to suggest and defend a different etiology. The study of beriberi is confined to more recent times, and, during the last twenty years, has received the attention of many capable investigators. The researches of EIJKMAN, GRIJNS, BRADDON, and FRASER and STANTON, among others, have proved beriberi among the rice-eating populations of the East Indies, where it is prevalent, to be due to a definite deficiency in the diet of the persons afflicted. The defect was traced to a loss of specially valuable constituents of the rice grain which occurred during milling and polishing after the modern method, and these workers showed that the disease could be prevented if unmilled rice were substituted for milled rice in the diet, or if the bran removed during milling were added to the polished rice. The later workers on this subject include SCHAUMANN (1910) and FUNK (1913), in whose papers a complete bibliography of the subject may be found. To FUNK (1912) we owe the expression "vitamine," which he invented to express the unknown essential principle whose absence from a diet occasions beriberi. The term has, however, become established in general use to signify any accessory substance necessary for satisfactory metabolism, deficiency of which in a diet will lead to the occurrence of a "deficiency" disease.

The present position may be summed up as follows: For perfect nutrition the human being requires—in addition to an adequate ration of fat, protein, carbohydrate, salts and water—a sufficient supply of accessory food factor or vitamines. These substances have not so far been successfully isolated; little is known of their chemical or physical properties, and, at the present time, their presence can only be detected by biological methods. There are, at least, two† distinct classes of these vitamines:

*JAMES LIND. "A Treatise on the Scurvy." 2nd ed. London. 1757.

† Rickets and pellagra, and some less known diseases of cattle, are also generally classed among the diseases due to defective nutrition: investigation in these cases is, however, not nearly so far advanced as in scurvy and beriberi.

(1) the vitamine whose presence in a diet is an essential for the proper nutrition of the nervous system, and whose absence or deficiency will give rise to beriberi—for the sake of brevity this may be called the anti-neuritic or anti-beriberi vitamine, and (2) the anti-scorbutic vitamine, whose absence or deficiency in a diet will occasion scurvy with its characteristic pathological changes.*

Scurvy and beriberi are now known to be respectively caused by a deficiency in these two different classes of vitamine, which possess different properties, play a different part in metabolism, and are differently distributed among food-stuffs in nature.

Deficiency diseases are practically non-existent among modern civilised Europeans, for, when living upon the ordinary mixed diet of civilisation, it is difficult to avoid getting an adequate amount of each separate vitamine. It is where the diet is more simple, as is the case with many Eastern races, that the risk of deficiency disease becomes proportionately greater. The food then needs careful scrutiny to ensure that all necessary vitamines should be included in the comparatively few substances of which the diet is composed.

In the case of Europeans, however, a variety of special causes may lead to what is virtually a restricted diet, *qua* vitamines, although it may not be apparent. Armies on active service, exploration parties in arctic regions, may enjoy a varied supply of preserved and sterilised foods but in effect be deprived of vitamines, these being wholly or in part destroyed by the high temperatures and other processes to which the food has been subjected in the course of preparation. Under such conditions beriberi or scurvy may take some months to develop to an acute stage, but there is little doubt that the defective diet will give rise to a general condition of ill health and inefficiency some time before definite symptoms of these diseases can be diagnosed by the physician. It is clearly of the utmost importance to ensure that a satisfactory diet should be available for soldiers and sailors on active service, more especially when one considers the great strain, both mental and physical,

*The spongy and septic condition of the gums characteristic of scurvy is probably partly, if not wholly, due to secondary bacterial infection. The defences against invasion are well known to be weaker in the mouth than in many other parts of the body, and in scurvy are further reduced owing to the abnormal condition of the blood system. This may account for the view frequently advanced, with some experimental support, that scurvy is an infectious disease of bacterial origin.

to which they are subjected. The first step towards this achievement is to obtain trustworthy information as to the distribution of these essential vitamines in ordinary foodstuffs. Some of this knowledge is already available, and it was with the hope of supplementing the existing data upon the subject that the present work was undertaken.

II. EXPERIMENTAL.

1. DISTRIBUTION OF ANTI-BERIBERI VITAMINE.

The distribution of the anti-beriberi vitamine among various foodstuffs was made the subject of a special investigation by COOPER (1913 and 1914), working in this Institute. We have used his methods, and our endeavour has been to extend his work with special reference to substances which are dry and portable, and hence specially suited to the rationing of troops on active service.

The deficiency polyneuritis of birds (which is readily induced experimentally by a diet of polished rice, see Fig. 1) has been accepted as analogous to human beriberi from the similarity of its etiology, symptoms and method of cure. COOPER chiefly used the pigeon as experimental animal. This bird is specially adapted for the study of the anti-neuritic vitamines, seeing that it is extremely susceptible to polyneuritis, but does not apparently take scurvy, even when deprived of anti-scorbutic material. Our work has also been done with the pigeon, and, following the methods of COOPER, we have studied the anti-neuritic properties of a series of foodstuffs by means of the following two types of experiments :—

A. Preventive Experiments.—In which the aim was to determine the minimum amount of the foodstuff that must be added to a vitamine-free diet to prevent onset of polyneuritis. Polished rice (40 grams daily) formed the vitamine-free diet, and prevention was reckoned to be successfully accomplished if the bird shewed no symptoms after 50-60 days. Unprotected birds usually develop acute polyneuritis after 15-25 days.

B. Curative Experiments.—In which the birds were fed upon polished rice until acute symptoms of polyneuritis were observed. Determination was then made of the minimum amount of the foodstuff in question which would effect a cure when administered by the mouth. Birds suffering from acute polyneuritis will usually die within twenty-four hours if not treated. In order to get the requisite amount of cura-

tive material (anti-beriberi vitamine) absorbed in time, it was frequently necessary to concentrate the vitamine. To do this a weighed quantity of the air-dry material was extracted with alcohol by shaking up with alcohol in the cold for some hours. The alcohol was then evaporated at low temperature under reduced pressure, and the residue was taken up in a small measured quantity of water. Definite amounts of this watery solution formed the curative doses, but in order to obtain a fairer comparison the amount of the dose was reckoned in terms of the original foodstuff.

TABLE I.

PREVENTIVE EXPERIMENTS.

Minimum daily ration which must be added to a diet of polished rice to prevent onset of Polyneuritis (Beriberi) in a pigeon of 300 to 400 grams weight.

SUBSTANCE.	DAILY RATION. Natural foodstuff. (Grms.)	DAILY RATION. Dry Weight. (Grms.)	OBSERVER.	NOTES ON RESULTS.
Wheat Germ, sample R.I., free from bran ..	1·5	1·3	H.C. & E.M.H	Complete protection.
Wheat bran, sample R I., free from germ	More than 1·5	More than 1·4	„ „	No protection with 1·5 gms. daily. Polyneuritis occurred in same time as in control birds.
„ „	More than 2·5	More than 2·2	„ „	Small degree of protection. Mean time of onset delayed about two weeks.
Yeast extract, commercial, sample A	0·5	0·35	„ „	Protection not secured in all cases.
	1·0	0 7	„ „	Protection.
Pressed yeast .	2·5	0 5	COOPER, *Journ. Hyg.* 1913 and 14	
Lentils	–	3·0	„ „	
Barley, unhusked	3·7	3·2	„ „	
Barley, husked	5·0	4·5	„ „	
Egg-yolk	3	1·5	„ „	
Beef-muscle...	20	5.0	„ „	
Ox heart-muscle	5	1·7	„ „	
Ox brain	6	1·2	„ „	
Ox liver	3	0·9	„ „	
Sheep brain...	12	2·5	„ „	
Fish-muscle...	More than 10	More than 2	„ „	
Cheese	More than 8	More than 5·6	„ „	Protection not secured with these amounts.
Cow's milk	„ „ 35	„ „ 3·5	„ „	

TABLE II.

CURATIVE EXPERIMENTS.

Minimum amounts of foodstuffs required to cure Polyneuritis (Beriberi) in a pigeon 300 to 400 grams weight. The doses are reckoned in terms of the original foodstuffs.

SUBSTANCE.	Preparation of curative material.	Amount of dose given. In terms of the natural foodstuff.	Amount of dose given. In terms of dry weight 100–110° C.	RESULT.	OBSERVER.
		grams.	grams.		
Wheat germ—					
Commercial sample, cooked .	Extracted with alcohol ...	8	6·9	Improvement ..	H.C. & E.M.H.
		12	10·3	Sometimes cures	,, ,,
		16	13·8	Cure	,, ,,
Sample A "picked," uncooked	Extracted with alcohol ..	10	8·7	Incomplete cure	,, ,,
		15	13·0	Complete cure	,, ,,
Sample B "picked," uncooked	Unextracted .	1·0	0·9	Sometimes cures	,, ,,
		2·5	2·2	Cure	,, ,,
*Maize germ	Unextracted ...	1·0 to 3·0	—	Cure . ..	,, ,,
*Rice germ	Unextracted ...	0·5 to 1·0	—	Cure	,, ,,
Wheat bran—					
Stone ground, not free from grm.	Unextracted	5·0		Sometimes cures	,, ,,
Roller milled, free from germ...	Unextracted ...	3·0		No cure	,, ,,
		5·0	—	Sometimes cures	,, ,,
Rice bran, containing germ ..	Unextracted ...	6·0		No cure	,, ,,
		10·0		Complete cure ..	,, ,,
Turbot fish-roe (hard)	Extracted with alcohol ...	35	10	Cure almost comp	,, ,,
		70	20	Cure complete ...	,, ,,
		140	40	Cure complete ...	,, ,,
Egg-yolk	Extracted with alcohol ...	60 = 4 yolks	30	Cure	COOPER, 1913.
Dried whole egg, com'l, samp. I.	Given without extraction ...	40 = abt. 4 ,,	38	Cure	H.C. & E.M.H.
,, ,, ,, ,,	Do.	30 = ,, 3 ,,	29	Cure	,, ,,
,, ,, ,, ,, II.	Do.	20 = ,, 2 ,,	—	Cure	,, ,,
Malt Extract—					
1st sample	Given without extraction ..	5	4·2	Cure	COOPER, 1914, I.
2nd sample	Do.	7	5·1	Cure	,, ,,
3rd sample	Do.	10	9·2	No Cure	,, ,,
Meat extract, com'l, sample I.	Do.	3	2·8	No improvement, bird died ...	H.C. & E.M.H.
,, ,, ,, ,, ..	Do.	6·5	5·2	No improvement, bird died ...	,, ,,
Raw beef	Extracted with alcohol ...	140	30 approx.	Cure	COOPER, 1913.
"Maconochie" ration	Do.	440	106	Cure incomplete	H.C. & E.M.H.
"New ration" roast beef tinned, submitted for examination by the Dept. of Hygiene, R.A.M. College, on 19th June, 1916 ..	Do.	350	112	Very slight improvem't, no cure	,, ,,

* Picked out from the un-milled grain by hand in the laboratory.

TABLE II. *continued.*

CURATIVE EXPERIMENTS—*continued.*

Minimum amounts of foodstuffs required to cure Polyneuritis (Beriberi) in a pigeon 300 to 400 grams weight. The doses are reckoned in terms of the original foodstuffs.

SUBSTANCE.	Preparation of curative material.	Amount of dose given. In terms of the natural foodstuff.	Amount of dose given. In terms of dry weight 100–110° C.	RESULT.	OBSERVER.
Dried peas	Extracted with alcohol	30	26	Incomplete cure	,, ,,
,, ,, ...	Extracted with alcohol	40	35	Complete cure	,, ,,
Pea flour, kilned .	Unextracted	10	9	Cure	,, ,,
	Extracted with alcohol	80	—	Slight impr'vm't No cure	,, ,,
Dried lentils	Do.	20	18	Cure	COOPER, 1913.
Dried vegetables, commercial	Do	40	35	Cure	H.C. & E.M.H.
Sample "Spring greens"	Do	120	105	Complete cure ..	,, ,,
Potatoes, peelings	Do.	180	36	Died	,, ,,
,, ,, .	Do.	630	126	Improvement, no cure	,, ,,
Potatoes, insides .	Do.	200	40	Incomplete cure	,, ,,
,, ,,	Do	350	70	Cure .	,, ,,
Pressed yeast	Autolysed	2·0	0·4	Cured slowly	,, ,,
,, ,,	Do.	3·0 to 6·0	0·6 to 1·2	Cure .	COOPER, 1914, II.
Yeast extract, com'l, Sample A	Given without extraction	1·5 to 2·0	1·0 to 1·3	Complete cure	H.C. & E.M.H.
Dried fruits, currant	Extracted with alcohol ...	60	47	No improvement	,, ,,
,, ,, ,,	Do.	19	16	No improvement	,, ,,
,, ,, dates .	Do.	26	21	Slight improv'nt, death delayed	,, ,,
,, ,, ,,	Do.	26	21	Slight temporary improvement	,, ,,
Proprietary article, dry powder, stated to contain vitamines in a concentrated form	Given without extraction	12 31		No improvt., died Temp. improvement, no cure	,, ,, ,, ,,

In case of cereals and some other materials rich in anti-neuritic vitamine, we gave the curative dose of the original foodstuff without preliminary extraction. By this means we avoided the loss of vitamine due to the process of extraction. This loss was very considerable; see, for example, the case of dry peas given in Table II., where the curative dose

of unextracted material was 10 grams, but that estimated from experiments made with an extract was as high as 40 grams. The original material was administered, without extraction, whenever possible, in order to obtain a better absolute value for the vitamine-content of the foodstuff in the natural condition. The drawback of this method is that absorption is slower and one does not get the swift dramatic cures commonly obtained when the curative principle is given in a soluble form. But if care is taken to see that the animal's crop is empty before administering a cure, digestion will not be too slow to permit of sufficient vitamine being absorbed in time to save the bird.

There is no doubt that the most trustworthy results are obtained from the preventive tests. They are, however, very exacting and laborious, the feeding is artificial, and must be continued for about two months, with daily careful supervision. If the enquiry were limited to this type of experiment its scope would be seriously restricted. In considering the values obtained from curative experiments, allowance must be made for a large margin of error, seeing that it is impossible to control the onset of polyneuritis so that the type of symptom and the severity of the condition should always be the same at the time of administration of the cure.

The principal data obtained are set forth in Tables I. and II., in which also many of the results of COOPER *(loc. cit.)* are included for the sake of completeness. Some of the results given in these Tables were anticipated by the workers mentioned on page 150, but COOPER was the first to make a systematic attempt to obtain a comparative set of values for the different foodstuffs as regards ability to prevent beriberi.

From Tables I. and II. it is seen that the anti-beriberi vitamine is extremely widespread, and is found present to a greater or less degree in almost every natural foodstuff investigated. The principal sources of this vitamine, however, are at once seen to be in the seeds of plants or the eggs of animals, where it is probably stored* as a provision for the nutrition of the offspring during the early period of its existence.

From the practical point of view, the most important result is the fact that *one of the chief sources of the anti-beriberi vitamine should be in the seeds of plants, including, as this does, the cereals and edible pulses.* The

* It is interesting in this connection to note that among a population upon the verge of beriberi owing to a defective diet, it is frequently the pregnant women who are the first to shew acute symptoms (see VEDDER. "Beriberi." London. 1913. p. 61).

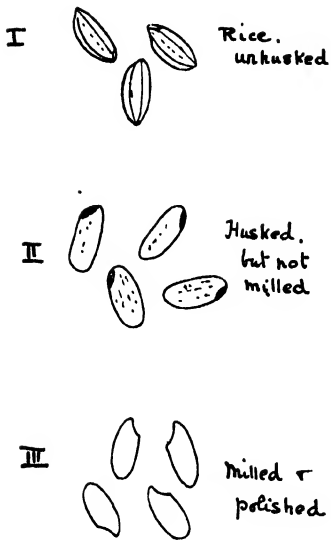


FIG. 1.

Shewing the various stages in Milling of the Rice Grain.

I. Rice grain in the natural condition, retaining the husk or enclosing glumes.

II. After removal of the husk, but retaining the pericarp or "silver-skin," and the embryo, which is shaded.

III. After milling and polishing; both "silver-skin" and embryo are removed and the grains are then "polished" by rubbing with talc between sheepskins.

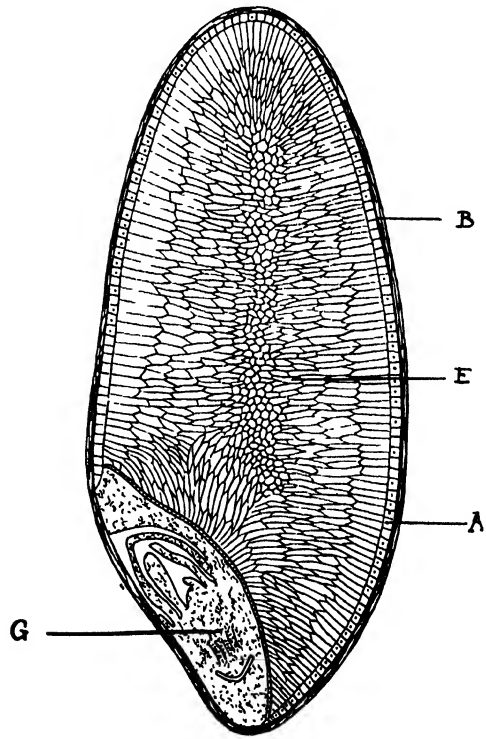


FIG. 2.*

Diagram of a longitudinal section through a grain of wheat, shewing

B Pericarp, forming the branny envelope.

A Aleurone layer of cells forming the outermost layer of the endosperm removed with the pericarp during milling

E Parenchymatous cells of the endosperm.

G Embryo or germ.

*Reproduced, with the permission of the Controller of H.M. Stationery Office, from Figs. 1 and 2 in Dr. J. M. HAMILL'S "Report to the Local Government Board on the nutritive value of bread made from different varieties of wheat flour," 1911.

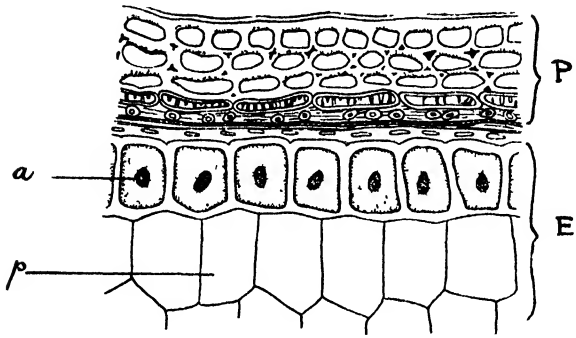


FIG. 3.*

Cross section through the branny envelope and outer portion of the endosperm of wheat grain, shewing—*P*, the pericarp; *E*, endosperm, consisting of *a*, layer of aleurone cells and *p*, parenchymatous cells.

*Reproduced, with the permission of the Controller of H M. Stationery Office, from Figs. 1 and 2 in Dr. J. M. HAMILL'S "Report to the Local Government Board on the nutritive value of bread made from different varieties of wheat flour," 1911.

great value of certain beans (*e.g.*, KATJANGIDJO, *Phaseolus radiatus*) in the prevention and cure of both avian polyneuritis and human beriberi, has been repeatedly shewn by GRIJNS (1901) and the other Dutch observers. In the case of cereals* an interesting differentiation exists in the different parts of the seed (see Figs. 1, 2 and 3). The largest deposit of the vitamine is present in the *embryo* or germ, while the bran (pericarp + *aleurone layer*) comes second in order of importance. The *endosperm* (especially when deprived of the *aleurone layer*, which is included in the bran in modern milling) is deficient in anti-neuritic vitamine, and will cause beriberi if employed as sole diet. One well-known example of a cereal endosperm is polished rice (see Fig. 1), where the germ is removed with bran during the milling and polishing. Another is ordinary white wheaten flour, in the preparation of which a similar deprivation takes place in the modern roller mills. In this case, during the "break" on the rollers, the oily germ is squeezed out flat, and subsequently can be separated completely from the flour and the bran. Both white flour and polished rice produce polyneuritis in pigeons in an exactly similar way, and, if the less complete evidence available in case of rye and maize be also taken into account, there is no doubt a general rule

*A more detailed account of our experiments with cereals is published in the *Proc. Roy. Soc.*, 1917, read February 15th, 1917 (in the press),

that among cereals the anti-neuritic vitamine is concentrated in the germ.

The potency of the cereal embryo in the cure of polyneuritis is very great. In case of wheat germ, 2·5 grams usually (and rarely 1·0 gram) sufficed to cure a pigeon of 300-400 grams' weight in an acutely ill condition. In case of rice embryo, cures were obtained with amounts varying from 0·5 gram to 1·0 gram.

The prevailing opinion, hitherto, has been that the substance preventing beriberi was situated in the cuticle of the husked grain, in the layer of cells *(aleurone layer)* immediately below the pericarp (see Fig. 3). This view is based upon the well-known influence of decorticated rice in inducing human beriberi, and the prevention and cure of the disease by the addition of rice polishings and their extracts. The true explanation lies in the fact that in the milling and polishing of rice the germ is removed with the bran (see Fig. 1). On comparing the values of (1) commercial rice bran and (2) the pure germ, separated from the grain by hand in the laboratory, we found the latter ten times as potent as the former for the cure of pigeon polyneuritis.

An interesting analogy with the seeds and embryos of plants is offered by the eggs of birds and fishes, which also form valuable sources of anti-beriberi vitamine. The successful cures obtained with desiccated eggs shew the great resistance offered by this vitamine to drying.

Yeast is another substance rich in anti-neuritic properties, which it retains even after extraction or autolysis. Yeast was the only unicellular organism investigated, and it may be significant of the universality of this vitamine that it should be abundantly present in this instance.

Milk, cheese and potatoes appear to be among the least valuable foods from this point of view; other vegetables and meat contain a moderate amount of anti-beriberi vitamine, but are very inferior when compared with eggs or the seeds of plants.

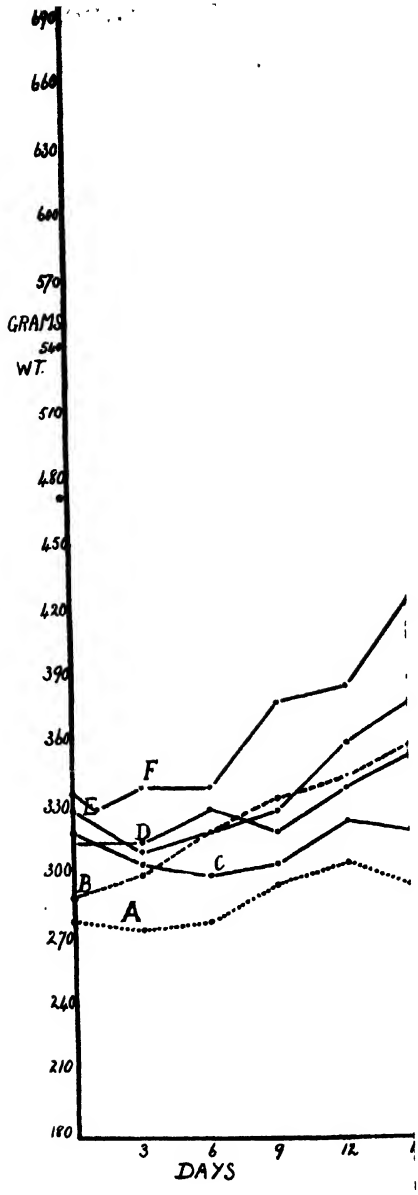
2. DISTRIBUTION OF ANTI-SCORBUTIC VITAMINE.

While there is a good deal of empirical knowledge upon this subject, few accurate scientific data are available, and what exist are found chiefly in the work of AXEL HOLST and his colleagues Drs. FÜRST and FRÖLICH, in the University of Christiania (1907, 1912, 1913). By depriving guinea-

pigs of fresh green food, and offering only a diet of grain and water, these workers were able to induce a disease analogous to human scurvy, from which the animal died within a month from the commencement of the restricted diet. The post-mortem appearances shewed all the lesions of typical scurvy, and included changes in bone, cartilage and bone marrow, loosening of the teeth, and, in addition, hæmorrhages, which might occur in any situation. These workers also studied the influence of various additions to this scurvy diet, and investigated the distribution of the anti-scorbutic principle among various vegetable foodstuffs and its resistance to drying and to exposure to high temperatures. The commonly accepted view that fresh vegetables and fruits are the chief source of anti-scorbutic vitamine was abundantly confirmed by their experimental work.

No symptoms suggesting beriberi were detected, nor were any nerve lesions discovered in these experimental animals, whose scurvy diet of various grains and water included unmilled cereals and contained abundant anti-neuritic vitamine. In any circumstances, the guinea-pig appears to be so highly susceptible to scurvy that attempts to produce beriberi have not been successful. The distribution of the anti-beriberi vitamine is very widespread, and on any diet adapted to this animal it seems impossible to protect satisfactorily from scurvy without at the same time supplying a sufficiency of the anti-neuritic substance. For this reason the guinea-pig is as admirably adapted for the study of scurvy as is the pigeon for that of beriberi.

During the last few months we have extended the work of HOLST and his colleagues in the direction of foodstuffs which are portable and convenient for transport, and hence suited for the rationing of armies. The methods we have followed have, in principle, been those of the Norwegian workers. The principal modification has been the addition of milk, subjected to prolonged heating at a high temperature (120° C. for one hour), to the scurvy diet of grain and water. By this addition we have not been able to detect any appreciable change in the special symptoms of the disease or time of onset; but the general condition of the animal is much improved, apart from the scurvy, and the influence of the inclusion of the anti-scorbutic substances can be studied with less complication from such factors as mere inanition and starvation (see Table III., and compare curves A and B in Fig. 4).



Weight Charts of five typica

CURVE A. Typical scurvy on a diet of

CURVE B. Typical scurvy on a diet of milk (heated to 120° C. fo

CURVE C. Typical scurvy on a diet of o by addition of orange juic the diet on the 22nd day,

The experiments have almost invariably been of the preventive type. There is no particular advantage in the curative type of experiment, where scurvy is concerned. The onset of the disease is gradual, and by the time the symptoms—loss of weight, painful joints and loosening teeth—are well marked, the lesions are extensive and the animal is in a very weak condition. If at this stage a powerful anti-scorbutic, such as orange juice, is given, the cure is extremely slow, and it may be many weeks before the animal may be said to be in perfect health. In fact, it is doubtful whether the joints ever become perfectly normal, and even when the cured animals seemed to have reached a thriving condition, one could frequently detect a slight tenderness of the arm and leg joints by a careful examination.

The general course of these experiments may be seen from Fig. 4 and Table III., which give weight charts and brief protocols respectively of three typical cases.

Experiment I. (Curve A, Fig. 4) is a case of typical scurvy on a diet of oats, bran and water. The animal (280 grams' weight), which is in a young and growing condition at the beginning of the experiment, puts on no weight upon this diet. After seventeen days distinct tenderness of the joints is present, and from this time the weight begins to decline. After about another ten days the teeth are found to be loose, little food is taken, the loss of weight becomes very rapid, and death soon follows. The post-mortem appearances generally found in such cases are as follows:—Extensive hæmorrhages are found present in the muscles of leg and thigh, especially near the knee-joints; less severe ones are frequently found in the muscles of the fore limbs, round the axilla. Subcutaneous hæmorrhages are also frequently present, although not to be detected during life, owing to the thickness and pigmented nature of the skin. The bones are fragile and easily break off at the junction of shaft and epiphysis; this is specially true in the case of the tibia, which is often found to be already fractured. The jaws are very brittle and the teeth usually quite loose. There are marked swellings on the ribs at the bone-cartilage junction, which is often found to be fractured. On histological examination these junctions are found to be much disorganised and shew the characteristic changes in the bone marrow, so carefully studied and described by Holst and Frölich (1912).

Experiment II. (Curve B, Fig. 4) shews very clearly the improved

condition when autoclaved milk is substituted for water in the diet, although the course of the scurvy is not modified. Weight is better maintained throughout, and, at the beginning of the experiment, some growth takes place.

Experiment III. (Curve C, Fig. 4) deals with a successful cure from scurvy by means of fresh orange juice. The first part of the experiment, on a diet of oats, bran and water, reproduces Experiment I. very closely. On the 22nd day, when the guinea-pig is losing weight rapidly and shews acutely sore joints, orange juice (10 c.c. daily) is added to the diet, and 50-60 grams of autoclaved milk. Little improvement is seen for four or five days, and the fall in weight continues. After this, however, a gradual improvement takes place, but it is 6-7 weeks after the beginning of the "cure" before the animal may be considered normal. The soreness of the joints persists long after the general health and weight have been restored.

The Curves D, E, and F, Fig. 4, shew the weight curves on diets which give adequate protection from scurvy, and are included for purposes of comparison. Curve E shews the increase of weight upon a normal diet of oats, bran and cabbage leaves (30 grams daily); and Curves D and F shew the result of substituting fresh orange juice for the cabbage leaves, with the addition also of autoclaved milk.

TABLE III.

EXPERIMENT I. (Guinea-pig 47). Diet: Oats, Bran and Water *ad lib.* (see Curve A, Fig. 4).

Date.	No. of days from beginning of Experiment.	Condition of the Animal.	Weight (Grams).	Amount of oats and bran eaten daily, average (Grams).
29-11-16	0		280	
16-12-16	17	Leg joints tender	290	(21 days) 34.
20-12-16	21		280	
25-12-16	26	Leg joints tender, incisor teeth loose	240	(9 days) 14.
29-12-16	30	Died	185	

EXPERIMENT II. (Guinea-pig 49). Diet: Oats, Bran and Autoclaved Milk (1 hour at 120° C.) *ad lib.* (see Curve B, Fig 4).

29-11-16	0		290	
15-12-16	16	One leg joint tender	350	(17 days) 25.
25-12-16	26	Teeth loose	240	
27-12-16	28	Died	230	(12 days) 5.

TABLE III.—*continued.*

EXPERIMENT III. (Guinea-pig 58). Diet: Oats, Bran and Water *ad lib.* for 22 days, with production of acute Scurvy; followed by the addition of Orange Juice, 10 c.c. daily, and Autoclaved Milk, about 50 grams daily, leading to a satisfactory cure (see Curve C, Fig. 4).

Date.	No of days from beginning of Experiment.	Condition of the Animal.	Weight (Grams).	Amount of oats and bran eaten daily average (Grams).
6-12-16	0		320	
16-12-16	10	One leg sore	315	
22-12-16	16	"Scurvy position" noticed, the sore hind-leg is raised in the air while the animal lies down on the other three legs.	320	(18 days) 33.
24-12-16	18	Great fall in weight begins	320	
28 12 16	22	Animal very weak *Cure started*, 10 c.c. fresh orange juice + 50-60 grms. autoclaved milk daily added to oats & bran diet	250	(5 days) 10.
29-12-16	23	Condition very bad	235	
1-1-17	26	Still very sore	235	
3-1 17	28	Lying in "Scurvy position"	270	(10 days) 15.
5-1-17	31	Soreness of joints improved	290	
7 1-17	33		295	
13-1-17	39	General condition good - joints still sore	325	
24-1-17	50	Ditto	365	
13-2-17	60	Soreness much improved	460	(41 days) 45.
21-2-17	68	No symptoms	482	
27-2-17	74	Excellent health, no symptoms	540	

It will be gathered that these experiments are laborious and tedious. The guinea-pigs in most cases require a certain amount of hand feeding to gain assurance that the requisite doses of the materials studied are really taken, and much care is also needed to render the foodstuffs palatable, for many of those studied are far removed from the natural food of this animal, and different treatment is required in each separate case. Further, a good deal of skilled attention is necessary if the general health and weight of the animal are to be maintained, apart from the question of scurvy. Under these circumstances the output of work possible from any one worker is limited, and we have been fortunate in obtaining the assistance of the following workers to whom, in conjunction with ourselves, the investigation of the following foodstuffs has been entrusted:—

Miss RUTH SKELTON (fresh and dried vegetables and lime juice).

Dr. E. Marion Delf and Miss Olive Lodge (fresh fruit juices; pulses, soaked and germinated).

Our endeavour is to obtain accurate quantitative figures expressing the relative anti-scorbutic value of a large range of different foodstuffs. The work is at present incomplete, but enough information is available from our own results and those of the Norwegian workers (confirmed throughout by our own) to construct the following Table IV., in which a rough estimate is given of the comparative value of the various substances in the prevention of *(a)* beriberi and *(b)* scurvy.

Table IV.

VALUE OF FOODSTUFFS AS PREVENTIVE AGAINST SCURVY AND BERIBERI.

Foodstuffs.	Water content per cent. (Approx.)	Value against Beriberi.	Value against Scurvy.
CEREALS -			
Whole grain, wheat	10 to 13	+ +	0
Endosperm, polished rice		0	0
,, white flour (wheat)			
Bran, *e.g.*, rice		+ +	0
,, ,, wheat			
Germ or embryo, *e.g.*, rice		+ + +	0
,, ,, ,, wheat			
PULSES—			
Whole, in dry condition	12	+ +	0
GERMINATED PULSES (or Cereals)	50	+ +	+ + +
VEGETABLES—			
Potatoes	80	0	+ +
Fresh, *e.g.*, cabbage	90	+	+ + +
,, ,, onions			
,, ,, carrot			
Desiccated vegetables	10 to 15	+	+ to 0 according to age
Pickled, *e.g.*, cabbage	--	—	0
FRUIT JUICE—			
Fresh, *e.g.* orange	90	—	+ + +
,, ,, lemon			
EGGS—			
Fresh	70	+ +	—
Desiccated	6	+ +	0
MEAT -			
Fresh	70	+	+
Tinned		0	0
MILK—			
Cow's, fresh	87	0	+ (slight)
YEAST—			
Pressed, autolysed	77	+ + +	0
Extract, commercial sample A	30	+ + +	0

— Signifies not investigated.

It is at once evident that the distribution of the anti-scurvy vitamine is much more restricted than that of the anti-beriberi vitamine. There are here no special places where deposits are found of highly concentrated anti-scorbutic material, but it is present in all living (actively metabolic) tissues of plants and (to a much less degree) in those of animals

The anti-scorbutic principle has not been found present in dried vegetables or in any dried seeds, such as cereals or pulses, diets of which form the classic means of producing the disease of scurvy. If, however, *these seeds are moistened and allowed to germinate, the anti-scorbutic principle is created anew with the beginnings of active cell life.* As far as our present knowledge goes, the presence of the anti-scorbutic vitamine is always associated with living tissues in which active metabolism is taking place When viable seeds are in the dry, resting condition, all the *active* processes concerned with life and metabolism are temporarily suspended, and a disappearance of the anti-scorbutic principle accompanies this cessation. If the seed is moistened, rapid

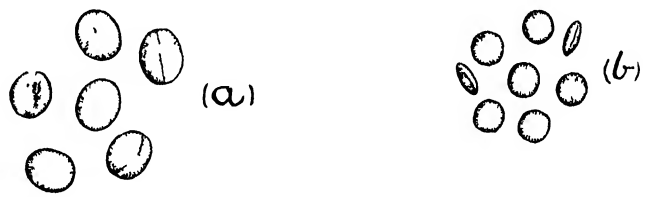


FIG. 5.

Pulses in the dry condition (natural size): *a*, Peas (*Pisum sativum*); *b*, Lentils (*Lens esculenta*).

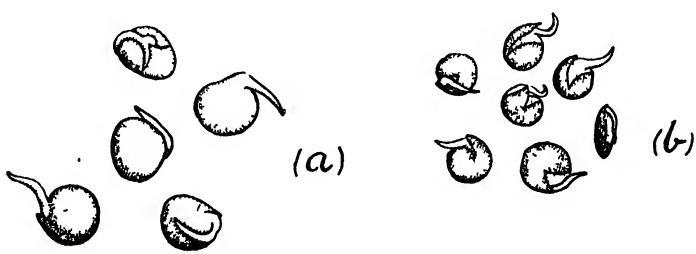


FIG. 6.

Same pulses, as in Fig. 5, after soaking in water for 24 hours and germination for 48 hours, at laboratory temperature (natural size): *a*, Peas (*Pisum sativum*); b, Lentils (*Lens esculenta*).

absorption of water takes place, the cells once more become turgid and the active processes associated with germination and growth will begin within a few hours. Accompanying this new activity the anti-scorbutic vitamine makes its appearance in the tissues and gradually increases in amount.

These facts were first discovered by FÜRST (1912), and have been amply confirmed by ourselves and our colleagues in an extensive set of experiments with peas *(Pisum sativum)* and lentils *(Lens esculenta)*, see Fig. 5. Our method has been to soak these seeds in water for 24 hours, during which time they absorb their own weight of water. They are then loosely packed in a glass funnel and maintained in a moist atmosphere for 1-2 days longer, care been taken to ensure abundant access of air. After 48 hours at laboratory temperature (50-60 F.) the radicle is usually from 0·5 to 1·0 c.m. in length (see Fig. 6), and at this stage we have used the germinated seeds in our experimental diets. Our work is not yet sufficiently advanced to permit of a final statement of the anti-scorbutic value of these germinated pulses in terms of fresh vegetables, but we have obtained complete protection from scurvy with a daily ration of 5 grams, which indicates a vitamine-content comparable with that, for example, of fresh cabbage leaves.

Fruit Juice.—Fresh fruit juices are exceedingly rich in anti-scorbutic vitamine. HOLST and his co-workers pointed out the value of fresh lemon and fresh raspberry juice in preventing experimental scurvy, and our researches have been made chiefly with orange and lemon juice. With these latter we have also been able to effect satisfactory cures from an acute scorbutic condition.

The results of our experiments with preserved fruit juice, *e.g.*, lime juice, have not been so encouraging, the anti-scorbutic power being very feeble in comparison with fresh juices. We are now investigating various methods of preservation in the hope that we may be able to suggest improvements. After reading the history of our Navy and Mercantile Marine, it is impossible to resist the conviction that, in former times, preserved fruit juice was of distinct value in preventing human scurvy, and it is not unlikely that modern methods of manufacture may have introduced some modification detrimental to the anti-scorbutic principle it originally contained.

Fresh Vegetables.—Among the fresh vegetables investigated, the cabbage was found best, partly, no doubt, because fresh green leaves happen to be the natural food of our experimental animal. Onions were also found to be powerfully anti-scorbutic, in spite of their dry appearance from the outside. Ordinary Spanish onions contain 95 per cent. of water, the cells are all in a turgid condition, and, botanically, the vegetable is to be regarded as a live bud. Potatoes and carrots must also be regarded as composed of live turgid tissue; they are also of great value, though found to be slightly inferior to cabbage leaves. In our experiments the potatoes were boiled before giving to the animals, and doubtless there was some loss of the anti-scorbutic principle during the process of cooking. It was, however, a necessary measure, as guinea-pigs are not able to take potatoes in the raw condition.

Milk.—Cow's milk possesses very low anti-scorbutic value. In our experience a ration of 50 grams daily was insufficient to protect guinea-pigs from scurvy. FRÖLICH (1912), however, found that they could be protected by an exclusive diet of milk, and we have found the same result if a minimum of 100 grams is taken daily. This is about twenty times the necessary daily ration (viz., 5 grams) of fresh cabbage or germinated peas, or fresh fruit juice. It seems probable that infants nourished on cow's milk do not obtain any excess of anti-scorbutic principle, especially if the milk is previously boiled. The modern custom of adding a small daily ration of fresh fruit juice, or other anti-scorbutic, appears to have sound and scientific foundation.

Meat.—It is practical experience, obtained especially from the history of Arctic exploration, that fresh meat will prevent human scurvy if taken regularly in fair quantity. The expressed juice of raw meat is also considered to be of value in the cure and prevention of infantile scurvy. For prevention of experimental guinea-pig scurvy, meat was found to be very disappointing, for little, if any, protection could be demonstrated. It must be remembered that meat is an unnatural food for guinea-pigs; they will not eat it even when cooked, and the experiments have to be made with the expressed juices. Allowance must also be made for the high susceptibility to scurvy of this animal, which is undoubtedly much greater than that of the human being; but, when all this is taken into account, one is forced to conclude that meat is far inferior as an anti-scorbutic to vegetables and fruit, and that the daily allowance must

be large if reliance is to be placed upon it for the prevention of scurvy (see also below, p. 171).

Yeast.—Yeast Extract A and autolysed yeast, both of which were shewn to contain the anti-beriberi vitamine in high concentration, uninjured by the process of preparation, were found useless for the prevention of scurvy. Animals receiving as large a daily ration as could conveniently be taken, died of acute scurvy in the same period as the control animals on the "scurvy diet."

RESISTANCE OF THE ANTI-BERIBERI AND ANTI-SCORBUTIC VITAMINE RESPECTIVELY WHEN EXPOSED TO DRYING, HIGH TEMPERATURES, ETC.

(A) Drying.—From the common presence of the anti-beriberi vitamine among dry foodstuffs, it is evident that this substance is not sensitive to the process of drying. It is very significant that its chief sources should be found in dry seeds, such as cereals and pulses.

The case is otherwise with the anti-scorbutic vitamine. This principle seems to be *absent or deficient in all dry foodstuffs*, such, for example, as cereals and pulses, and to be rapidly destroyed when the animal and vegetable tissues, in which it is naturally contained, are subjected to drying. HOLST and FRÖLICH *(loc. cit.)* found dried vegetables of little use for the protection of guinea-pigs from scurvy, and we have confirmed their observations. A similar result has been noted in human experience. Dried vegetables were found useless in the epidemic of scurvy which ravaged the Austrian army in Hungary in the early part of the 18th century (BUDD, 1840), and, including dried potatoes, were tried with the same disappointing results during the American Civil War ("Medical History of the War of Rebellion," Vol. III., Washington).

The temperature at which the tissues are dried seems to be a matter of indifference. In HOLST and FRÖLICH'S experimental work (1912) low temperatures were employed, not exceeding 37° C. The destruction of the anti-scorbutic principle during and after drying, appears to be a gradual process of spontaneous decomposition, which follows disorganisation of the living cells in association with which it is produced. If the tissues are rapidly brought to an absolutely dry condition, this process of destruction is checked and the anti-scorbutic properties are preserved to

a much greater degree (see HOLST and FRÖLICH, 1913). In a similar way these workers found that the expressed juices of acid fruits shewed more stability in respect of their anti-scorbutic value than those of fresh vegetables, and concluded that the rate of this spontaneous decomposition of the vitamine was slowed by the presence of an acid medium. This affords some scientific foundation for the general belief that preserved fruit juices may be relied upon to retain some, at least, of their original anti-scorbutic value.

(B) High Temperatures.—There are many isolated observations upon the influence of exposure to high temperatures upon the anti-beriberi vitamine, but no one hitherto has attempted any systematic study of this point. GRIJNS (1901) found that 1-2 hours' exposure to 120° C. destroyed the protective powers of unmilled rice, Katjangidjo beans *(Phascolus radiatus)*, and buffalo meat against avian polyneuritis. EIJKMAN (1906) and SCHAUMANN (1910) confirmed GRIJNS' results on the whole, though the former observer found a greater resistance present in the case of horseflesh. HOLST (1907) detected no particular loss of anti-neuritic vitamine after exposure of dried peas and unpeeled barley to 115° C. for half an hour, but some damage in case of beef after half an hour at 110° C. Most observers have failed to find any significant destruction of this vitamine at 100° C.

The discrepancy between the results of these various experiments is to be explained partly by the roughness of the biological test for the presence of the vitamine in question. It is also partly due to the fact that certain substances originally containing a low concentration of this vitamine (*e.g.*, meat) may be unable to protect from or cure polyneuritis after a certain degree of exposure to high temperatures, whereas others originally richer in this material (*e.g.*, pulses, cereals, etc.) may, after a similar exposure, yet retain a sufficient amount to afford satisfactory protection or cure. Another point to be considered in these experiments is that the temperatures noted appear to be those registered by the autoclave or steamer in which the heating was done, and that no measurements are recorded of the temperatures in the interior of the substances heated. This is an important point, as the latter temperatures remain for a long time below the former, the degree of difference depending on the conductivity of the material heated, and will vary with every type of substance investigated. It therefore seemed to us worth while to make a

series of systematic experiments on this point, and a brief résumé of the results obtained* is set forth in Table V.

Two substances were chosen for the investigation, both rich in anti-beriberi vitamine, viz. *(a)* the germ *(embryo)* of wheat, and *(b)* yeast Extract A.

TABLE V.

INFLUENCE OF EXPOSURE to high temperatures upon the anti-beriberi vitamine contained (*a*) in Wheat Embryo and (*b*) in Yeast Extract.

SUBSTANCE	Temperature. C.	Time. Min.	Minimum amount required to cure a pigeon (300 to 400 grms.) suffering from acute Polyneuritis. (Grams).
Wheat embryo	unheated	Control	1·0 to 2·5
Water content, 11 to 14 %	98 to 103	120	2·5
,, ,, ,,	100 to 117	40	5·0
,, ,, ,,	118 to 124	120	10·0, did not cure
Yeast extract, A—	unheated	Control	1·5 to 2·0
Water content, 30 %	100	60	2·0 to 3·0
,, ,, ,,	122	60	2·5 to 3.0
,, ,, ,,	122	120	about 5.

The data in columns 2 and 3, as to the temperatures reached and the times of exposure, refer to the internal temperatures of the material heated, and were compiled in accordance with the results of a special set of control experiments made for the purpose. The vitamine content of the different samples was estimated by determining the minimum amount necessary to cure acute polyneuritis of pigeons, brought on by an exclusive diet of polished rice. The results in Table V. shew that the anti-neuritic properties of wheat-germ and yeast extract are only slowly impaired by prolonged heating at or near 100° C, but that destruction is much more rapid at temperatures in the neighbourhood of 120° C.

The experiments at 100° C. were devised to be comparable with the ordinary processes of cooking, while those at temperatures from 110° C. to 120° C. were arranged to throw light upon the probable fate of anti-neuritic (anti-beriberi) vitamines during the sterilisation of tinned foods,

* This work is given in detail with full protocols of the individual experiments in a special communication upon the subject—see *Proc. Roy. Soc.*, *B*, 1917, read February 15th, 1917 (in the press).

such as tinned meat, etc. In this connection it is interesting to compare the curative values given in Table II. of—

(a) Raw beef, where the curative dose was an extract equivalent to 30 grams dry weight of the original, and

(b) "Maconachie" ration and "New Army" ration, where an "incomplete cure" and "no cure" were obtained with extracts equivalent to 106 and 112 grams dry weight, respectively.

The destructive influence of high temperatures upon the anti-beriberi vitamine is also seen by noting the superior curative properties of dried peas (unheated) compared with "kilned" pea flour.*

As was the case in exposure to drying, so in exposure to high temperatures, the anti-scorbutic vitamine appears to be much more unstable than the anti-beriberi vitamine. There exists no systematic experimental work upon this point, and we are now starting a series of experiments with the hope of filling the gap. There are, however, some valuable observations of HOLST and FRÖLICH (1912), all of which point to the great sensitiveness of this vitamine to high temperatures. When cabbage leaves were boiled at 100° C., the results obtained indicate that at least one half its anti-scorbutic value was lost in 30 to 60 minutes, and after heating to 120° C. for 60 minutes almost *all* power of protection against scurvy was destroyed (*loc. cit.* Table IV., B p. 70).

In the light of these results it is not to be expected that the anti-scorbutic vitamine will survive the heating to which tinned foods are subjected in order to render them sterile.

III. PRACTICAL APPLICATION OF THE EXPERIMENTAL WORK TO THE PREVENTION, RESPECTIVELY, OF HUMAN BERIBERI, AND HUMAN SCURVY.

The practical use that can be made of these investigations on the distribution of the two classes of essential vitamines will depend on the identity of avian polyneuritis and human beriberi on the one hand, and of guinea-pig scurvy and human scurvy on the other. As regards etiology, nature of symptoms and method of cure, the analogy in both

* This fact needs emphasis, since many samples of pea flour on the market are kilned in the process of preparation, although no mention of this fact is made on the wrapper, etc.

cases is so perfect that they can be considered as physiological equivalents, and the information thus acquired can be applied forthwith.

There is, however, in addition, a great deal of direct evidence to be extracted from human experience. The individual instances may not seem to be altogether conclusive, but when taken together they form a mass of evidence which is very impressive, and afford a striking confirmation of the results obtained from experimental work. To exhaust these instances would need a very long communication. We propose, however, to describe briefly a few cases which seem to us among the more instructive.

PREVENTION OF BERIBERI.

Importance of the nature of the Cereal food in the prevention of human beriberi.—In almost all cases human beriberi is to be attributed to a defect in character of the cereal employed in the diet.

The best known and best studied case is that of the rice-eating populations of the Dutch Indies, Malay States, the Philippine Islands, etc.: it has been the subject of numerous series of researches, among which those of EIJKMAN, GRIJNS, BRADDON, FRASER and STANTON, VEDDER and CHAMBERLAIN are among the more important. All these workers are agreed that the disease is caused by a defect in the rice taken, which defect can be traced to the complete decortication which takes place in the modern steam-milling of the grain. Beriberi is prevented in cases where unmilled rice is substituted for the "polished" rice, or where the bran ("polishings") removed in the milling is added to it. The general opinion is that the anti-beriberi vitamine is contained in the inmost layer of cells *(aleurone layer)* of the skin, which layer of cells is removed with the bran in the process of preparation. Our experiments indicate that the *germ (or embryo) of the grain, also removed during milling, is the principle source of the anti-neuritis vitamine,* present also to a less extent in the bran (see Table II.). It is not so generally realised, however, that the same principle applies to other cereals, or that an equal danger of beriberi is to be apprehended for a wheat-eating population under certain circumstances. In the modern "roller"-milling of wheat, there is complete separation of bran and germ from the flour: unless, therefore, these constituents are purposely included later (as in brown flour, and "standard" flour) one may regard ordinary white flour

and the bread or biscuit baked from it, as being free from these valuable constituents.

The deficiency of anti-neuritic vitamine in white wheaten flour was shewn by HOLST (1907), EDIE and SIMPSON (1911) and also by ourselves (see footnote page 150), to occasion polyneuritis in pigeons in a manner exactly similar to polished rice. The following three incidents shew that these results are also applicable to the case of beriberi in man. Under ordinary circumstances this deficiency of vitamine in white bread is well supplied in the other articles of the usual mixed diet of the European. It is in cases where extremes of climate restrict the variety of available foodstuffs, or where there is temporary separation from fresh food supplies on long sea voyages, or by the exigencies of active service on long campaigns, that this defect in the bread ration may become apparent in a tragic manner.

(1) LITTLE (1912) states that in Newfoundland and Labrador, where in mid-winter and spring many persons are obliged to subsist largely on bread, beriberi frequently occurs. At the present time the bread is made from fine wheat flour; in the memory of the older inhabitants, when the bread was made from "brown" flour, the disease was unknown. In 1910 the following interesting event took place:—A ship laden with whole-wheat flour ran ashore, and a considerable proportion of her cargo was removed in order to lighten her, and later was consumed by the adjacent population. There was no case of beriberi in that region for a year following this occurrence.

(2) Beriberi was rare on Norwegian ships before 1894, after which date it became much more frequent. This frequency coincided with an alteration of diet which was made compulsory in that year in response to a popular demand for an "amelioration" of the conditions of life in the Norwegian mercantile marine. Previously the sailors on long voyages used biscuit made from rye flour; subsequently the masters of ships were obliged to supply bread baked from white wheaten flour, or a mixture of wheat and rye flour (HOLST, 1911). It is an interesting corollary upon our own experimental work to note that *in the milling of rye flour there is no separation of the germ.*

(3) The most impressive case of all is to be found in the tragic experiences of our own troops recently operating in Mesopotamia. In his "Account of the Medical Arrangements, etc., during the Siege of

Kut-al-Amara" (Dec. 1915 to April, 1916),* Colonel HEHIR, I.M.S. (1917), states, "in the early stage of the siege a recrudescence of beriberi amonst British troops gave rise to some apprehension, *but it then disappeared;* whilst in Indian troops and followers during the latter half of the siege scurvy caused anxiety." From other entries in this vivid and valuable diary, it is seen that the British troops in the garrison received a cereal ration of wheat flour during the first two months of the siege. After February 5th, 1916, from one-third to one-half of this flour was replaced by barley flour and by "atta," the coarsely-milled wheat usual in the Indian sepoy's ration. It is very significant that beriberi should have broken out among the British troops while upon their normal ration of white wheaten flour, and should have cleared up when they were obliged to share in the more coarsely milled (and doubtless germ-containing) grain of their Indian fellow-soldiers. There is no doubt, from the wealth of detail given in Colonel HEHIR'S report, that the British troops were protected from scurvy by the ample rations of meat† (12oz. at first; later, Jan. 22nd, 1916, 8oz.), or horseflesh (1¼lbs. March 4th, 1916), served out to them throughout the siege. The Indian soldiers, while protected from beriberi by the nature of their cereal ration, failed, in many cases, to obtain a sufficient supply of anti-scorbutic vitamine, owing to their refusal to eat fresh meat, in spite of the admirable and persuasive manifesto issued by Colonel HEHIR upon the subject.

During the whole operations in Mesopotamia the Indian soldier seems to have been well protected from beriberi. This was to be expected, for, in addition to an unspoilt cereal, he normally gets a generous daily ration of "dhall," consisting of various dry pulses, which, as may be seen from the foregoing work, are also valuable sources of anti-beriberi vitamine. These, however, did not afford protection from scurvy, which was specially prevalent among the Indians, as, for example, in the summer of 1916, when, presumably, great difficulty was experienced in providing fresh fruit and vegetables *(Mesopotamia Report, X. 42).*

* Appendix III. Mesopotamia Commission Report, 1917.

† This meat appears to have been fresh, except possibly between dates of Dec. 30th and January 29th. Fresh meat has some protective power against beriberi, but this is slight compared with that of grain and pulses. It would be of great interest to know whether, during the outbreaks of beriberi among British troops, prior to the siege of Kut (Colonel HEHIR speaks of a "recrudescence" during the siege) the men were receiving tinned or fresh meat.

The deduction to be drawn from all this experience is as follows:—

For the prevention of beriberi it is in the highest degree desirable that the germ (embryo) and the bran of wheat should not be excluded from the flour destined for manufacture of bread and biscuit for troops on active service. This is the more necessary when the troops are separated from fresh food supplies and the rest of the ration consists largely of tinned foods, seeing that these articles are deficient in* all *vitamines, owing to their previous sterilisation at high temperatures.*

Yeast and Yeast Extract. -Yeast is acknowledged to be a most valuable source of anti-neuritic vitamine There is not much evidence at present available as to the value of yeast and yeast extracts in the prevention of human beriberi; we have not been able to find any report of such a trial. Ordinary dried yeast is very disagreeable to take, and causes digestive disturbance, but many preparations possess none of these defects. They have the agreeable savoury taste of a meat extract, and are largely used as an ingredient in certain soup cubes on the market. We have examined one of the principal commercial yeast extracts (Yeast extract A, Tables I. and II.), and could detect no loss of the original vitamine of the yeast owing to the method of preparation. Soup squares form at all times a very popular addition to the soldier's ration, and might well be limited to those containing a certain proportion of yeast extract, seeing that pure meat extracts have been found deficient in anti-beriberi vitamine (see Table II.). By such a measure a valuable extra supply of anti-beriberi vitamine could be added to the ration in a convenient and palatable form.

Dried Eggs.—It will be seen from the examination of two commercial samples of desiccated eggs given in Table II., that so valuable an article of diet as the fresh egg can be successfully dried without losing the anti-beriberi properties. Dried eggs may prove too expensive to form any part of the ordinary soldier's or sailor's dietary, but there can be no doubt as to their suitability for inclusion in hospital stores. Intestinal infections form a large part of the medical casualties in armies on active service, especially when operating in hot climates, and, during both the acute

* In packing and transporting whole-meal or germ-containing flour, extra precautions are usually considered necessary to protect from damp, and the advice of experts should be sought upon these points. No possible difficulty could arise in the case of biscuit baked before transport. Certain samples have been specially investigated by us and were found to be entirely satisfactory for the prevention of beriberi.

stage and the convalescence, the diet is usually restricted to tinned milk and invalid foods, all of which may be regarded as vitamine-free. Mention is made below of the possibility that intestinal affections may in some cases prove a predisposing cause of deficiency disease. It is, therefore, highly desirable that all supply of vitamine should not be cut off during such an illness. Fresh eggs, which provide anti-neuritic vitamine and are also well suited for an invalid diet, are seldom obtainable, and we would, therefore, strongly recommend that dried eggs should be freely used as a substitute. Both commercial samples examined by us were found to be very rich in anti-beriberi vitamine, they were soluble and could be taken raw, and, when cooked, they made excellent and very palatable dishes.

PREVENTION OF SCURVY.

It is a platitude that scurvy can be prevented by the inclusion of fresh vegetables and fruit in a dietary. These are frequently impossible to obtain under the conditions of active service. It is, therefore, our intention under this heading to treat only of such substitutes as are convenient for transport, and hence suited to the needs of armies in the field.

Germinated Pulses as a Substitute for Fresh Vegetables.—The most important fact that has emerged from the study of experimental scurvy is the discovery of FÜRST (1912) that the anti-scorbutic vitamine, absent or deficient in the resting seed, makes its appearance in the early stages of germination. In our own experiments with peas and lentils, these were first soaked in water for 24 hours, the temperature ranging from 50° to 60° F; at this stage a small amount of anti-scorbutic vitamine could already be detected. After germination for a further 48 hours, with access of air, the vitamine-content had increased five- to six-fold, and at this stage the germinated material may be considered the equal of fresh vegetables as regards its anti-scorbutic value (see Table IV. above).

It is difficult to imagine any circumstances in which this form of anti-scorbutic food could not be made available. In the dry form, peas, beans, lentils and other pulses contain only 10 to 15 per cent. of water, and are eminently suited for transport. They are commonly included in the active service dietaries of armies, and in the case of Indian troops, as "dhall," form a considerable proportion of the daily ration. The only

change needed is that these pulses should be issued in the unmilled (not husked or split) condition, and should retain the original seed-coat. Then, in case of unavoidable shortage of fresh fruit or vegetables, a substitute could be made immediately available on the spot by germinating the pulses included in the stores.

The whole operation consists of a preliminary soaking, in which the seeds absorb over 100 per cent. of their original weight of water, followed by germination; after this the food should be cooked and eaten as soon as possible. The time taken by the complete operation could be reduced to 36 hours in a hot climate. In Appendix I. below, p. 178, a set of directions is given, which may be found a useful guide for preparing these germinated pulses in the field.

It is very important that the peas, lentils, etc., should be cooked and eaten as soon as possible after germination, and should not be allowed to dry again. In the process of drying, the anti-scorbutic vitamine developing during the germination, will be destroyed. Having regard to the sensitiveness of this vitamine to high temperatures, it is also important that the pulses should not be cooked longer than is necessary to render them soft and palatable. A period not exceeding 1 to 1½ hours should suffice for peas and about half an hour for lentils. No deterioration in flavour can be detected after germination has taken place.

Dry pulses contain a high proportion of anti-beriberi vitamine, and this was found by GRIJNS* (1901) to be lessened in amount as germination takes place.

We found that in the *early* stages of germination an abundant supply of anti-beriberi vitamine was still retained by our peas and lentils. Dramatic cures of pigeons with acute polyneuritis were obtained with moderate doses (5 to 10 grams) of peas and lentils germinated to the stage, shewn in Fig. 6, in which condition we were using them successfully for the prevention of scurvy.

* Captain COOK, whose long voyage (1772-1775) was notable for the continued good health of the sailors, always took with him a large supply of malted barley. He had the greatest belief in the anti-scorbutic value of a freshly made infusion (sweetwort) and served it out to his men in case of need ("Captain Cook's Voyages," *Everyman* Edition, p. 227).

Some Chinese are accustomed to take part of their daily rice in the germinated condition, and this custom has spread to the Malay States (private communication from Brig.-Gen. ANDERSON) and Dutch Indies (GRIJNS, 1901), where "towgay" or germinated pulses are regularly taken. There appears to be no evidence, however, that the anti-scorbutic properties of these foods have been appreciated.

Freshly germinated pulses (or cereals), therefore, occupy a special position among foodstuffs in being richly endowed with both the anti-scurvy and the anti-beriberi vitamine.*

Vegetables.—Among vegetables the onion is marked out as being specially suited to the need of troops, owing to its great resistance to adverse conditions during transport and to the length of time it will remain wholesome. The anti-scorbutic value is comparable with that of fresh cabbage and its flavour gives it more value as a culinary adjunct than is possessed by any other vegetable

Potatoes were found to be somewhat inferior in anti-scorbutic properties to cabbage, onions, etc. They are more suitable for transport and keep better than most vegetables, and there is no doubt that they have been proved of great value for the prevention of human scurvy. It must be remembered, however, that the amounts consumed are rather large. In one Irish workhouse† the daily ration of potatoes is 3 lbs.; after a careful study of convict diets, Dr. GUY‡ concluded that 14oz. daily would protect from scurvy, the rest of the ration including 1oz. of other fresh vegetable and 4oz. of meat. Outbreaks of scurvy have repeatedly followed failure of the potato harvest in countries where potatoes are a staple article of diet, *e.g.*, Norway in 1904 (HOLST, FRÖLICH, 1912) and Ireland in 1847 (CURRAN). The recent appearance of scurvy in Glasgow§ and Newcastle‖ and Manchester is no doubt to be attributed to the great scarcity of potatoes during the last three or four months.

Fresh Meat.—The expressed juices of fresh meat gave disappointing results in the prevention of guinea-pig scurvy, but there is no doubt that human beings can be protected by its regular use. This is one of the cases which suggest that the guinea-pig is much more susceptible to scurvy than is man, an opinion also held by HOLST and FRÖLICH (1912). Many clinicians believe fresh meat juice to be capable of curing infantile scurvy, and in many Arctic expeditions where scurvy has been escaped, fresh meat of various kinds has been the only anti-scorbutic material present in the diet.

* Refers to note on previous page.

† Limavady Union, Co. Derry.

‡ Dr. GUY'S evidence, Report of the Lords Commissioners of the Admiralty on the Outbreak of Scurvy in the recent Arctic Expedition. 1877, par. 5323.

§ *Lancet*, July 8th, 1917.

‖ HARLAN. *British Medical Journal*. July 14, 1917.

The following incidents are related by JACKSON and HARLEY (1900:— Six Russian priests spent the winter in a hut at Kharborova, Yugor Straits, with a small Russian boy to wait upon them. Their religion would not permit them to eat reindeer or such meat; they subsisted largely on salted fish, and there were no vegetables. In the following May the little Russian boy was found to be the only surviving person in Kharborava; he was subject to no religious restriction and ate reindeer meat during the winter. JACKSON himself, living among the Samoyads in Waigatz, 1893-1894, noted that among those of the population who winter on the island, and get neither vegetables nor lime-juice, but live largely on fresh reindeer meat, scurvy is unknown. Some Samoyads, however, migrate with Russian peasant traders, and spend the winter near the large rivers in north-east Russia where the diet consists largely of salt fish; among these scurvy is prevalent.

There is no doubt, however, that animal tissues are distinctly inferior in anti-scorbutic properties to those of fresh fruit or vegetables, and that a large and regular ration is necessary for safety. This opinion was expressed by many witnesses in the Admiralty Enquiry* upon the outbreak of scurvy in the Arctic Expedition of 1875, and in the older literature many instances occur in which an apparently liberal meat ration did not prevent scurvy. CURRAN (1847) describes three cases admitted to Swift's Hospital, Dublin, in the great Irish epidemic of 1847, where the previous diet had included ¾lb. on five days of the week. Colonel HEHIR†, in a report suggesting needed reforms in the Indians soldiers' diet in Mesopotamia as early as April, 1915, expresses great doubt whether the authorised meat ration of 28oz. weekly is sufficient to prevent scurvy unless more meat is added and the vegetable ration (2oz. potatoes) is increased. If dry weight is taken into account in comparing the anti-scorbutic value of meat and vegetables, 4oz. meat must be regarded as the equivalent of at least 10oz. vegetables. The British troops during the siege of Kut-al-Amara were doubtless protected from scurvy by their meat or horseflesh rations, but these were very abundant (8oz. to 20oz. daily), see above, p. 165.

Milk.—Fresh cow's milk was found to possess very low anti-scorbutic

* "Report of the Lords Commissioners of the Admiralty upon the Outbreak of Scurvy in the Recent Arctic Expedition." London, 1877.

† Mesopotamia Report, 1917. X. 41.

value in feeding guinea-pigs; satisfactory protection from scurvy was only attained by a large ration, about 100 c.c. daily. It is probable that infants living upon cow's milk do not receive any great excess of vitamine. As regards adults, CURRAN (1847) instances upwards of 80 cases admitted to the Dublin Union Hospitals in the Irish epidemic of 1847; all had been inmates of the union for at least six months previously, and the diet, though deficient in vegetables and meat, had included at least one pint of milk daily.

We have, nevertheless, been much impressed with the great value of milk, even when heated to 120° C. for one hour to destroy the vitamines, as an adjunct to diet. The general condition of animals who received a minimum amount of anti-scorbutic material could be enormously improved by the addition of a daily ration of this heated milk, although the scurvy was not influenced. Therefore, while admitting that milk is of little importance for the prevention of scurvy (or beriberi), its inclusion in a diet, even when tinned and sterilised, would appear to be a very valuable measure.

Fruit.—Fresh fruit juices appear to be among the most valuable anti-scorbutic materials we possess. In our experimental work fresh orange and lemon juices were found very potent. There is also abundant evidence, dating as far back as the beginning of the seventeenth century,* of their value in the prevention of human scurvy. In 1795, largely due to the efforts of Sir GILBERT BLANE (1785, 1830), a regular issue of lemon juice† was ordered in the Navy, and a remarkable decrease of the death-rate was the result. BUDD (1840) states that, whereas 1,457 cases of scurvy were admitted to the Royal Naval Hospital at Haslar in the year 1780, no case was reported in the years 1803 to 1810.

The juice of limes has also been widely esteemed in the past as an anti-scorbutic. There is not, however, much trustworthy evidence as to the anti-scorbutic value of lime-juice when preserved according to modern methods. We have so far found it disappointing for the prevention or cure of guinea-pig scurvy. HOLST and FRÖLICH (1912, p. 96) on the other hand, demonstrated distinct anti-scorbutic properties in two commercial samples of lime-juice, purchased in retail shops in Christiania. The subject is one of very great importance, and we are at present

* JAMES LIND. "A Treatise on Scurvy," 2nd Ed. London, 1757.

† In 1840, the daily ration of lemon juice was 1oz. and 1½oz. sugar, issued two weeks after leaving land. (BUDD, 1840).

engaged on a further investigation, with special reference to the processes of preservation. In the meantime we are not able to express any definite opinion. One thing, however, seems very clear. There is no reason at all why preserved lime-juice should be regarded as a specially concentrated form of anti-scorbutic. This has seemed to be the view generally held, if one may judge by the small size of the ration issued. If lime-juice is issued at all as an anti-scorbutic the ration should be a liberal one—at least 1oz. daily.

IV. RELATION BETWEEN BERIBERI AND SCURVY AS REGARDS TIME OF ONSET, ETC.

It is evident that a diet defective in both classes of vitamine will occasion both beriberi and scurvy, but it is likely that beriberi will precede scurvy in point of time. In "ship" beriberi the symptoms are reminiscent of scurvy, and it is probable that there has been in the diet a deficiency both of anti-beriberi and anti-scurvy vitamines. The "period of resistance," or "period of development," will depend on the degree of deficiency and upon the idiosyncracy of the individual, but, on the whole, seems to be shorter in case of beriberi. There is good evidence upon this point in the observations of FRASER and STANTON (1909) who found beriberi developing among Javanese coolies after about 80 to 90 days upon a diet composed largely of polished rice. In case of scurvy, the time seems to be longer. Colonel HEHIR* considers four months to be the minimum time in which scurvy will appear in Indian troops on active service, and HOLST and FRÖLICH (1912) refer to two instances in which the time was longer. The first is that of a fanatical vegetarian who wished to prove to the world that human life could be sustained upon a diet of bread and water. The bread was presumably rye bread, and the individual therefore well supplied with anti-beriberi vitamine; he developed scurvy in 7½ months. The second was related by a political refugee in Norway, and deals with the case of 1,400 prisoners in a Russian convict prison. The diet consisted of tea, coarse bread and cabbage soup; the preparation of the latter was so unclean and disgusting that twenty of the prisoners, who were of gentler birth than the others, could not endure to take it. After about six months these twenty shewed symptoms of scurvy, while no case occurred among the rest of the prisoners who took the soup. This incident also shews that, even

* Mesopotamia Report, 1917, X., 41.

after a prolonged boiling, enough anti-scorbutic vitamine remained undestroyed in the cabbage leaves to protect from scurvy.

It is not improbable that the times of onset of these two deficiency diseases might be much shortened in case of any intestinal infection occurring during the "period of development." Such a disease might well impose a drain upon the metabolism which would involve the reserve of vitamines as well as the other resources. It is interesting in this connection to note that among twenty-six cases of beriberi diagnosed in our troops in the Dardanelles in the autumn of 1915, and noted by WILLCOX (1916), eighteen had suffered from diseases of the digestive and alimentary system, previous to the beriberi. At the same time it is possible that the first illness may only indirectly have predisposed to the beriberi by making it necessary to further restrict the already limited diet to such vitamine-free articles as tinned and sterilised milk, etc.

The following case (for details of which I am indebted to Dr. FRANK SAVERY, of Ealing) of a little girl, aged two, who acquired scurvy while suffering from chronic colitis is also to the point. Cow's milk could not be taken and the child was nourished on a brand of dried milk, with the addition of raw meat-juice to her diet as an anti-scorbutic. After some months on this diet, scurvy was diagnosed, but was readily cured by fresh orange juice. Fresh meat-juice has been considered by many clinicians an adequate anti-scorbutic for infants upon a diet of sterilised milk, and is also much esteemed as a cure for infantile scurvy. This incident suggests that the colitis rendered the child more susceptible to scurvy than a normal infant, and also points to the great anti-scorbutic potency of fresh orange juice in comparison with meat-juice.

SUMMARY.

1. To maintain a human being in a satisfactory state of nutrition the diet must contain—

   (a) A suitably proportioned supply of protein, fat, carbohydrate, salts and water, and

   (b) An adequate amount of accessory food factors, or vitamine.

Both *(a)* and *(b)* are required; excess of one cannot make good any deficiency in the other.

2. These necessary vitamines are, at least, of two kinds: firstly, the anti-neuritic or anti-beriberi vitamine, deficiency of which in a diet occa-

sions beriberi; and, secondly, the anti-scorbutic vitamine, deficiency of which occasions scurvy.

3. Neither of these vitamines has yet been isolated in a pure state, and, for the moment, their presence can only be detected by biological methods.

4. These two classes of vitamines have each their individual rôle in metabolism; they possess properties different from each other and are differently distributed among natural foodstuffs.

5. The distribution of the anti-beriberi vitamine has been investigated by study of experimental polyneuritis in birds, which is generally accepted as analogous to human beriberi. Pigeons, if deprived of anti-beriberi vitamine (*e.g.*, on an exclusive diet of polished rice or white flour, etc.), develop acute polyneuritis (beriberi) in fifteen to twenty-five days. The presence and relative amount of the anti-neuritic vitamine contained in various foodstuffs has been determined by means of curative experiments, and by preventive trials with specially selected diets. In this work we have extended the observations of COOPER (1913-14), and have, in general, followed his methods.

6. The anti-beriberi or anti-neuritic vitamine was found in almost every natural foodstuff examined. The principal source is in the seeds of plants, *e.g.*, cereals and pulses. The most important result emerging from the present work is the fact that *in cereals the anti-beriberi vitamine is mainly deposited in the germ or embryo of the grain, and to a less extent in the bran.* White wheaten flour or polished rice, which consist of the *endosperm* (minus *aleurone layer*) of the grain are deficient in this vitamine, and if employed as sole diet will occasion polyneuritis in pigeons or beriberi in man.

Other important sources of anti-neuritic vitamine are the eggs of animals (*e.g.*, hens' eggs or fish roe) and yeast or yeast extract. Milk and cheese gave disappointing results (COOPER, 1914).

7. From the abundant presence of the anti-beriberi vitamine in dry foodstuffs it is clear that this substance is resistant to drying. It can also withstand exposure to temperatures in the neighbourhood of 100° C. for two hours without significant loss; if heated under pressure to, or near, 120° C. destruction takes place more rapidly (see Table V.).

8. The distribution of the anti-scorbutic vitamine has been investigated by a study of experimental scurvy in guinea-pigs. We have

followed the methods of HOLST and his co-workers (1912) with some modifications. A diet of cereals with water or sterilised milk will cause death from acute scurvy in these animals within a month. The influence of various foodstuffs in preventing scurvy when added to this "scurvy diet" has indicated where the principal sources of the anti-scorbutic vitamine are to be found.

9. The anti-scorbutic vitamine is present in active living vegetable tissues. It is also present in animal tissue, to a much less degree. Fresh vegetables and fresh fruit juices are the most valuable sources of anti-scorbutic vitamine that we possess. All the dried foodstuffs examined, including desiccated vegetables, were more or less deficient in this vitamine.

10. Dry pulses (or cereals) though rich in anti-beriberi vitamine are deficient in anti-scorbutic vitamine, and afford no protection against scurvy. If these are moistened, however, and allowed to germinate, the anti-scorbutic principle is regenerated with the beginnings of active cell life.

Germinated pulses are recommended as a valuable and convenient means of preventing scurvy in the absence of fresh fruit and vegetables. In the dry stage they are eminently suitable for transport and can be moistened and germinated on the spot as required. In Appendix I. is given a method for preparing germinated lentils or peas under active conditions.

11. It is evident from 9 and 10 that the anti-scorbutic vitamine is extremely sensitive to drying. As regards exposure to high temperatures, it is also more unstable than the anti-beriberi vitamine. HOLST (1912) found that the anti-scorbutic power of fresh cabbage was slightly lessened after exposure to 100° C. for half an hour, but that the deterioration was appreciable after one hour; at 110-120° C. the destruction was rapid and complete.

12. It follows from 7 and 11 that neither the anti-neuritic vitamine nor the anti-scorbutic vitamine may be expected to survive in tinned or sterilised foods, considering the high temperature to which these have been subjected in course of preparation.

13. In case of armies or other populations subsisting largely on tinned food, it is imperative to provide adequate supply of vitamine from outside sources.

To prevent beriberi, the bread or biscuit should be made from wholemeal or germ-containing flour.

To prevent scurvy, if a supply of fresh fruit or vegetables is not procurable, germinated pulses should be added to the diet.

In conclusion, our best thanks are due to the following firms for generous help in the supply of material for our investigations to:—Messrs. Steele & Co., rice millers; Messrs. Brown & Polson, maize millers; The Hovis-Bread Flour Co. and Messrs. J. & H. Robinson, wheat millers, and especially to Mr. E. G. Ellis, of the last-named firm for much kind help, information and advice; Messrs. L. Rose & Co., for samples of specially prepared lime-juice; Elizabeth Lazenby & Son, Harrod's Stores, and Messrs. Crosse & Blackwell. We also wish to express our thanks to Professor HARDEN, F.R.S., for preparing some of the extracts mentioned in Table II.; to Miss MABEL RHODES, for kindly making the drawings from which figures 1 to 6 are made; and lastly to our assistant, Mr. A. H. ROBINS, for his devoted care and feeding of the experimental birds in the beriberi enquiry.

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APPENDIX I.

SUGGESTED METHOD OF USING PEAS, LENTILS, OR OTHER PULSES (DHALL) FOR THE PREVENTION OF SCURVY, IN THE ABSENCE OF FRESH VEGETABLES.

1. The dry seeds must be whole, retaining the original seed-coat, not milled or decorticated. See Fig. 5 (*a*) peas and (*b*) lentils.

2. They must be soaked in water for several hours; the time necessary depends on the temperature, 24 hours at 50° to 60° F. and 12 hours or less at 90° F.

3. The water must then be drained away and the peas, etc., allowed to remain in the moist condition with access of air. They will then germinate and the small rootlet grow out as in Fig. 6 (*a*) peas and (*b*) lentils. This germination will take 48 hours at 50° to 60° F. and 12 to 24 hours at 80° F.

4. The operations described in 2 and 3 could conveniently be done under active service conditions in such manner as the following :-

Soaking. -The peas or other pulses, placed in a *clean* sack, should be steeped in a trough, barrel, or other suitable vessel, full of clean water, and should be occasionally stirred. The sack and trough, etc., should be large enough to allow for the swelling of the peas to about three times their original size. In a hot climate 6 to 12 hours should suffice for this soaking

Germination. The peas should be lifted out of the water and spread out to a *depth not exceeding 2 to 3 inches* in a trough or other vessel with sides and bottom porous or well perforated with holes. This is to allow *complete* access of air. *The seeds must be kept in a moist atmosphere,* This is done by covering with damp cloth or sacking, which is sprinkled (by hand or automatically) as often as is required to keep the peas thoroughly moist underneath. The germinaiion should reach the stage shewn in Fig. 6 within 24 hours in a hot climate. *All the vessels should be clean.*

5. *It is important that the germinated pulses should be cooked and eaten as soon as possible after germination, and should not be allowed to become dry again;* in that case the anti-scorbutic properties, acquired during the process of germination, will again be destroyed. The pulses should not be cooked longer than necessary.

LISTER INSTITUTE,

June 15th, 1917.

Professor A. E. BOYCOTT: All I have to say is to draw the attention of the meeting to three microscopical specimens which are under the microscopes. Through the kindness of Miss CHICK, I have had the opportunity of examining a number of animals dead of this disease. I express no opinion as to whether it is the same condition as human scurvy, but the specimens in the room illustrate what is the only considerable anatomical change which one finds in these animals, namely, the disorder there is at the growing ends of bones. That is the lesion to which HOLST drew particular attention, and he used it as a diagnostic test as to whether an animal had experimental scurvy or not. It is always present in the guinea-pigs, and it consists of a number of complicated details, which are best appreciated by looking at the specimens. But, roughly, what happens, as far as we can make out, is that the preparatory changes in the cartilage, before it is turned into bone, do not come off, or if they do, they occur in some irregular way which obstructs the connective tissue, and prevents it performing its ordinary function. The result is, that at the growing ends of bones—and it is particularly well seen at the junction of the bony to the cartilaginous parts of the ribs—instead of there being an orderly conversion of cartilage into bone, there is disorder. The end of the medullary cavity, instead of being active tissue, forming periosteal bone and being filled with bone-marrow, is filled up with a lot of connective tissue which does not go on to the formation of bone. That wad of connective tissue is what gives the gross appearance to which HOLST draws attention particularly: that at the ends of these bones there is a clear area.

With regard to the rest of the organs of these animals, it is curious to note the extraordinarily little change one finds. The animals are very thin, wasted, etc.; but we have examined in a series of animals the various organs of the body without finding any change of any importance. There is no question that these changes in the bones are not what causes death; they are the dramatic and obvious anatomical peculiarity.

Under the microscopes there are two sections of the ribs of scurvy animals, and, for comparison, there is a section from a normal guinea-pig.

Sir PATRICK MANSON: I would like to congratulate the Society upon having received a lecture of so interesting a character. I have

seen a good deal of beriberi, from a clinical point of view, and I recognise that, in certain instances, the multiple peripheral neuritis which we call beriberi is the result of a deficiency in the matter of feeding. But I have a strong suspicion that that is not the explanation of all of those cases which we call beriberi. I remember very well a case of what looked like ordinary beriberi which developed in hospital; the patient died. I called it "beriberi;" it had all the signs of multiple peripheral neuritis, such as we usually mean by that word. After that patient's body had been removed, a patient suffering from stone in the bladder occupied the same bed. He developed the same kind of condition and died. I had crushed the stone, and the patient seemed to be doing well, but he developed multiple peripheral neuritis of the beriberi type and died. A very few days after that, a patient who had Potts' disease of the spine, occupied the same bed, and within a day or two, this patient also got multiple peripheral neuritis; and within a bed or two on either side similar cases also occurred. This strongly suggested to my mind that beriberi might be really an infectious disease, one which was transferable from one patient to another. Early impressions are strong, and this experience I have not forgotten. We are apt to be too much fascinated by a new theory in pathology, or even by words or phrases. There is a difficulty in explaining some disease; then some new pathological fashion turns up, and we are apt to say that it applies to the unexplained disease. Thus I fear that the term "deficiency disease" is prone to obsess our judgment in these cases of endemic or epidemic multiple peripheral neuritis. I have no doubt that a large number of cases of what we call "beriberi" are really etiologically different diseases. In epidemics among coolies in tin mines and in plantations, where the food is bad, badly-cooked rice, or over-milled rice, I think the disease is a deficiency disease. But I am convinced that there are other forms of epidemic multiple peripheral neuritis which have not received enough attention. I look upon the term "beriberi" as a word which refers to a group of symptoms, not to one pathological entity merely. I remember very well, some years ago, at the Seamen's Hospital, a patient came in with the usual signs of beriberi. There were plenty of beriberi cases in the wards at the time. He had difficulty in walking, œdema, palpitations, and all the usual signs of beriberi. On examining his blood, we found the malaria parasite. We gave him

quinine, and all the symptoms cleared up rapidly. I think that something similar must occur in many other infections.

I would like to ask the authors if experiments have been done by them with boiled rice in the case of pigeons, fowls, and guinea-pigs, and if so, what was the result?

Lieut.-Colonel J. C. MARTIN, F.R.S.: I was delighted to hear what Sir PATRICK MANSON said about Dr. CHICK'S account which we have just listened to, which seemed to me to be a model of exposition, and one suited to this time of day.

I have seen a good many hundreds of nutritional experiments, and, amongst them, the latter part of this research which Miss CHICK and Miss HUME have been conducting. And, first of all, I would emphasise the enormous amount of work which is involved in this kind of study. The chemist, to test the result of his experiments, puts a piece of litmus paper or other indicator in and gets his answer straight off; but here the answer to every question asked involves one or two months' careful watching and feeding of the animals. The result of one series of experiments must emerge before you know how to go on to the next step. Of all the nutritional experiments I have seen, these are, far and away, the best. The one thing which is fatal to nutritional work is, to send the animals away to an animal-house to be looked after by somebody else, and the great feature of this present work is, that these animals have been tended and nursed and fed by the observers themselves. It has always been known that they did actually take their ration, both their main ration, and the particular substance the effect of which it was hoped to ascertain.

Recently, there has, I understand, been a magnificent experiment on deficiency diseases made upon the garrison of Kut. Both beriberi and scurvy occurred amongst the garrison, the former in Europeans, the latter in Indians. As far as I know they did not overlap. The diets of the two kinds of troops were different. The Europeans had fresh horse-flesh and white bread and white flour. The natives had lentils and a certain amount of wholemeal flour, as, with the exception of the Ghurka regiments, they would not eat fresh horse. Those who had the fresh meat were saved from scurvy; whereas the native who would not eat the horse got scurvy. The native did not get beriberi because the

bulk of his diet consisted of pulses and coarsely ground attar (wheat).

The question of the exact pathology of the disease produced in the guinea-pig by depriving it of green food is interesting. HOLST'S results have been criticised on the grounds that the disease is not true scurvy. To my mind, it does not matter a bit whether it is so or not. The pathological condition, whatever its precise nature, is caused by depriving the animal of green stuff. I think that it closely resembles scurvy, not so much scurvy as seen in the adult, but rather more that of the baby, and it must be remembered that the animals used for this research were growing guinea-pigs. In adult scurvy the characteristic feature is the extraordinary tendency to hæmorrhages, and in older guinea-pigs deprived of green stuffs hæmorrhages are even more extensive.

Sir PATRICK MANSON asked Miss CHICK the question, whether the same polyneuritis was produced if the rice were cooked? I should be sorry to deprive her of the pleasure of answering it, but I am wondering whether she will remember that the discovery by EIJKMAN in 1897, that deficiency in diet was a cause of polyneuritis was actually made with boiled rice. EIJKMAN was the pathologist at a hospital in Java. For purposes of economy, the fowls were fed upon the rice that the patients left, and suffered from a curious disease, with paralysis, from which they died. EIJKMAN was led to enquire into the causation of this calamity to the fowls and found therein the interpretation of beriberi.

Mr. JAMES CANTLIE: In reference to what Sir PATRICK MANSON has said, I beg to state that my first introduction to beriberi was in Hong Kong, when taking charge of Sir PATRICK MANSON'S ward while he was on holiday. There were in the ward a number of surgical and a number of medical cases—two of them with beriberi. When Sir PATRICK returned, I pointed out to him that every man (six) in the ward who had an open sore or wound had contracted beriberi during his absence, while those men who suffered from ailments, with no sore or wound escaped infection. I became impressed at that time with the idea that beriberi was an infectious disease, inasmuch as six cases in that ward had evidently become infected after admission by the beriberi cases already in the ward. It is very difficult to get rid of an early impression of this nature, and with that experience before me I have always found it difficult to

subscribe whole-heartedly to the conclusion that beriberi is to be ascribed exclusively to aberrations in diet. I thought it worth while mentioning this fact in view of what Sir PATRICK MANSON has just said, as any observation by him always deserves the closest attention.

Lieut.-Colonel COPEMAN, M.D., F.R.S.: I would like to associate myself with what Sir PATRICK MANSON has said in returning thanks to Miss CHICK and Miss HUME for this very interesting investigation, in which I am especially interested, because being at present in charge of the Hygiene Department of the Royal Army Medical College, I have seen a good deal of the work in the course of its progress, and have, as a matter of fact, supplied some of the material, including Army lime-juice, which, apparently, was found to be absolutely useless for the particular purpose of scurvy prevention. Much of this work, as Miss CHICK said, has been done for the Army Medical Department, with the view of finding, in the first instance, some material which, while useful in the prophylaxis and treatment of beriberi, could be easily transported and would not readily undergo change under tropical conditions. I do not know whether Colonel MARTIN saw the "savoury tablets" we sent out before he came back. Miss CHICK tested them and found them useful in curing the polyneuritis of pigeons; some thousands of pounds have been sent out, but we have yet to learn what has been the result of their use on the human subject. They consist mainly of marmite, with a basis of pea-flour cooked in bacon-fat, to improve the flavour and to obviate the bitter taste which raw pea-flour possesses. I have recently heard from my colleague, Dr. BUCHANAN, in Mesopotamia, that beriberi is not so prevalent as formerly, but I would not like to say it is in consequence of the use of this material which has been sent out. Yeast is one of the best antidotes for the polyneuritis of pigeons, but even pressed yeast does not keep at a high temperature, especially in the presence of moisture. The tablets of which I speak have the advantage of not undergoing change in their appearance, their consistency, or their curative effect, after some considerable time, and after exposure to such temperatures as are experienced in the tropics.

With regard to the use of lime-juice in scurvy, I suppose some difference in value as an anti-scorbutic may be due to the fact that apparently the lime-juice manufactured now is sterilised or preserved

to a greater extent than previously. I do not know whether its diminished efficiency is due to added alcohol, or to the heat used in sterilising, unfavourably affecting the vitamines. But since Miss CHICK has demonstrated the fact that germinating peas contain anti-scurvy vitamines, these can be carried about in the dry condition, and one can subsequently cause them to germinate whenever and wherever you may want to use them.

The guinea-pig exhibited seems, so far as I know, to be in the condition described by Colonel MARTIN; the pathological condition being very similar to that seen in the scurvy of infants. But I agree with him it does not much matter what the precise condition is, so long as one gets the index required for experimental purposes.

I am very glad to have had the opportunity of hearing this lecture and seeing the demonstrations.

Fleet-Surgeon P. W. BASSETT-SMITH, R.N.: Lime-juice as supplied to Navy undoubtedly was useful in preventing scurvy. That lime-juice comes from the West Indies; it has a good body, is sweet, and differs from that so often supplied at the present time. Perhaps it is the superiority of the West Indian lime-juice which gives it its curative powers.

Dr. G. C. Low: I would like to say a few words on another branch of the subject, which has not been much touched upon in the paper, namely, the clinical aspect of beriberi. Large numbers of cases of this disease are constantly passing through the Royal Albert Dock Hospital, London School of Tropical Medicine, and since the question of vitamines has come prominently to the front we have been trying foods specially rich in these substances for curative purposes. We do not, however, find that the cases respond in the same marvellous manner as pigeons, and, as a matter of fact, it is rather difficult to be certain that they get better more quickly on those foods as compared with ordinary diet. Yeast has been tried, but here again the results are no better than before. It seems that in man the nerves take much longer to regenerate, and the changes produced are sometimes so extreme that the patient may lie ill for months irrespective of what sort of food is given him. I recollect one case who was shewing no signs of improvement, and he was given potato

skins with remarkable results; from that day he went steadily on and convalesced in a satisfactory manner. It was supposed that the vitamines in the skin were responsible, but after hearing Miss CHICK's paper this is not so clear, as I notice she says there are practically no vitamines in potatoes. It is possible, of course, as Sir PATRICK MANSON suggests, that all cases of multiple peripheral neuritis are not beriberi in the strictest sense, and other causes may be responsible. One could hardly expect vitamines to do much good in such cases. There is a large field open for further experimental work in the treatment of human cases of beriberi. Vitamines, I admit, will prevent such cases, but, once having occurred, what part do these substances play in accelerating the return of the nerves to normal?

The CHAIRMAN: Dr. LOW has just told us the remarkable effect of feeding a beriberi patient on potato skins, and would have us believe that it was due to the vitamines contained in them. But from my experience of military hospitals— and I am sure Fleet-Surgeon BASSETT-SMITH will bear me out— if we fed our men on potato skins we would almost empty our hospitals within twenty-four hours, whatever disease our patients were nominally suffering from.

You will agree with me that we have listened to a most interesting, instructive and important paper by Miss CHICK. What can be more important for a nation than its food? It is a subject which should receive much more attention than it does.

Miss H. CHICK: I think there is little to say in reply. It is not all pigeons which get cured so quickly. If you do not treat a case at the right moment, but let it go on, or give an insufficient amount to cure, so that there is a relapse in a few days, the disease may become chronic, and then the bird may take a month or six weeks to cure. And it must be remembered that all processes in the pigeon go on very quickly compared with the rate in the human. A patient who has had beriberi for some time, so that it has become chronic, will naturally take some time to cure. But in the case of ordinary acute epidemic beriberi, the cures are pretty quick when the right sort of diet is provided on the spot.

Dr. G. C. Low: The patients get it coming home on the ships. Many of our cases are quite acute; I was not speaking of old chronic cases.

Miss H. CHICK: I should like to know what would happen if one gave a diet with wheat germ in it to such cases, because we found in chronic pigeon cases which would not get better, that an occasional big meal of wheat germ improved them. Has anything like that been tried?

Dr. G. C. Low: No; but I shall be glad to try it in the next cases we have.

Proceedings of a Meeting of the Society held on Friday, June 15th, 1917, at 5.30 p.m., at 11, Chandos Street, Cavendish Square, W. 1, Sir RONALD ROSS, K.C.B., F.R.S. (*Hon. Vice-President*), in the Chair.

ANNUAL REPORT.

The CHAIRMAN called upon the Hon. Secretary to read the Annual Report and the Treasurer's Statement of Accounts.

Dr. LOW presented these statements in abstract, also the result of the ballot for President and Members of Council for next Session.

These the meeting agreed to.

The CHAIRMAN: Gentlemen, in the absence of our President, up till now Dr. SANDWITH, I have been called upon to induct into the Chair our new President, Sir DAVID BRUCE, whom, personally, I am delighted to see in this distinguished office. We must all remember, and we do all remember minutely, the work of Surgeon-General Sir DAVID BRUCE. It begins with the work that he did in Malta on Malta fever; and that was one of the most perfect bits of bacteriological investigation ever done, for he shewed clearly that *Micrococcus melitensis* is the cause of Malta fever. Next, you may remember his most distinguished work on nagana, about 1894. He was one of the very first to shew how insects are engaged in the transference of animal parasites from host to host. Then came his work, a little later, upon sleeping sickness. His Commission found the cause of sleeping sickness and the mode of its transference by means of a Glossina. Then he was President of a Committee of the Royal Society, supported by Government, to investigate Malta fever still further, with the result, as we all know, that it was shewn to be transferable largely through the agency of goats.

Sir DAVID BRUCE has been labouring consistently and constantly all his life on these great pathological problems, and the solution of any one of the problems I have mentioned is enough to give a man the greatest

credit in the world. I am sure that not only is the medical profession indebted to him for the work he has done, but the whole world is equally indebted And I need scarcely point out that a man who discovers the cause of a disease, or the mode of transference of a disease, confers a benefit on the whole of humanity—a benefit which will continue to be of use not only in the present but for all future time

I will now end these brief words of appreciation of our new President by inviting him to take the Chair

Sir DAVID BRUCE, C B , took the Chair

The PRESIDENT I have to thank you, gentlemen, for the very kind way in which you have received my being made President I have to announce that Mr J CANTLIE has been elected Treasurer Will anyone propose Auditors for the year?

Dr A G BAGSHAWE I propose that Dr. BEDDOES be re-elected and that Dr LEIPER be asked to take my place

Sir RONALD ROSS I second those proposals

The proposals were carried.

The PRESIDENT This settles the business at the annual meeting We now proceed to the papers

a.

YAWS. Before inoculation

b.

YAWS. One month after inoculation with "914"

III *c*

Yaws Before inoculation

III*f*

Yaws Two weeks after inoculation with 914

III

Yaws—Severe case

IV

Yaws—Typical papules on cheek

V

GANGOSA — Ngulunga or Mbaki

To accompany Father Gregoire's paper

VI

Mboko (?) Syphilis

TREATMENT OF YAWS AND OTHER DISEASES IN THE BELGIAN CONGO BY NEOSALVARSAN.

BY

FATHER G. GREGGIO, *Belgian Congo.*

— —

"914" (neosalvarsan) has been largely used among the tribes of the Inkissi Valley in the Moyen Congo Belge. About 500 negroes have been inoculated in less than three years at the Kisantu Lazaret, and, except in some exceptionally serious cases, the inoculations have been completely effectual.

The names the natives give the most important diseases treated are as follows:—(1) Nkoulou (Pian), (2) Nzeku or Bilwa, (3) Ngulunga-Mbaki, (4) Mboko.

1. Nkoulou (Pian), or yaws, is the most widespread infection. In Kingombe, a village belonging to the Kisantu Chefferie, out of 109 natives examined in 1911, fifty were found to have been infected during some part of their lives.

The natives acknowledge that the nkoulou is very easily contracted, As they are wont to say: "Let the child not sleep with his mother when she is infected with the nkoulou. . . . . Let her give immediately her child the manioc loaf she has bought. If the bread remains some time in her house it will become contaminated, and the child will get the nkoulou."

It is said that through manioc bread a tribe living on the right bank of the Inkissi was infected by the populations living on the opposite bank. These natives sold with their products manioc loaves in which they had hidden small particles of yaws crusts. They were convinced that by so exchanging the disease they were adopting the best means for ridding themselves of it.

This belief—the so-called "emigration of the nkoulou"—is said still to be held, but I have never been able to substantiate this for myself.

The natives have, however, another treatment for the nkoulou. Taking some pieces of forge scoriæ they make them incandescent, and with these extremely hot cinders they slightly touch the pian boils. Needless to say, this painful treatment has to be repeated from time to time. In some other villages the natives practise another kind or variety of treatment. The fetish-man finely grinds some scoriæ; he then adds water, soot, rifle powder, and tobacco previously rolled and crushed in his hands. With this composite powder he mixes some juice extracted from palm branches, lemon-squash, and finally fresh hen's dung. Then he warms the whole mixture, washes the patient, and removes the crusts in such a way that the blood begins to flow. Lastly, the remedy is applied in a burning condition to the bleeding wounds. This has to be repeated every three days.

Such long and painful cures help to explain the great success and popularity that "914" has achieved in this region of the Congo.

2. Similar good results are obtained when treating the nzeku or bilwa. By these names the negroes mean splittings of the skin which appear on the ankle or the heel or on the palm of the hand. Such lesions are never found elsewhere. As a matter of fact they are only terminal results of yaws. Some of these splittings or stripes last several years, and the natives admit that they are extremely painful.

3. Ngulunga-Mbaki.* Photo. V. gives a striking example of this disease at its highest pitch. It begins by a perforation of the palatal vault, and from the hole a somewhat greenish matter flows. Infected people are easily distinguished by their nasal accent. As I have experienced myself several times, the infection can be cured by means of the "914" if the treatment is applied at the beginning of the disease.

The patient I have photographed has been inoculated with 0·75 gm. of "914," but I do not know if he got better. Such a horrible infection is described by the natives, according to their superstitious belief, as a punishment inflicted upon those who steal anything in fields enchanted by the sorcerer.

4. The Mboko* causes large wounds amongst the natives, these emitting a very foul smell of rotten flesh, the distinctive mark of this

* Evidently gangosa.—ED.

* Probably syphilis.—ED.

infection. Photo. VI. shews clearly such lesions. The boy was inoculated twice with 0·45 gm. of the "914," with an interval of ten days between the two inoculations, and was completely cured.

DESCRIPTION OF PHOTOGRAPHS.

PIAN (YAWS).

I. Bisengo.
   (*a*) Before the inoculation. Inoculated once with 0·9 of "914."
   (*b*) One month after inoculation.

II. Kyangi.
   (*a*) Before inoculation. Received one injection of 0.9 of "914."
   (*b*) Two weeks after inoculation. He got completely cured.

III. Ntoto.
   The photograph shews the yaws papules at their highest development. Patient was completely cured by one inoculation of 0·9 of "914."

IV. Songia.
   Two characteristic papules on cheek.

V. Nyulunga or Mbaki.

VI. Mboko.

DISCUSSION.

Major ALDO CASTELLANI shewed by means of the epidiascope photographs of cases of yaws before and after treatment :—

(1) A very bad case of yaws in a Cingalese woman.
(2) The result ten days after treatment by means of tartar emetic, potassium iodide, sodium salicylate, bicarbonate of soda, and chloroform water.
(3) A photograph, belonging to Dr. SPAAR, of a case treated with the same mixture, before the treatment.
(4) The same case twelve days from the commencement of the treatment.

He continued: In my experience salvarsan, potassium iodide, and tartar emetic are the three drugs which give the best results in the treatment of yaws. As regards mercury, I would like to say at once that it has practically no influence whatever on the malady. Salvarsan certainly has the most rapid effect, but there are two drawbacks in connection with it. The first is that, in certain parts of the tropics, it is difficult to obtain; and the second is that it has to be given by intravenous injection, which means that it needs the services

of a medical man. In certain parts of the tropics hospitals are scarce, well-qualified medical men are rare, and treatment is in the hands of native assistants, or even native nurses. Under these circumstances, I think, internal treatment by the mouth is very useful. The mixture, which I devised some years ago, has yielded me good results. It contains: Tartar emetic, ½ to 1 gr.; potassium iodide, 1 drachm; sodium salicylate, 10 grs.; bicarbonate of soda, 15 grs.; water or chloroform water to 1 oz. This mixture has been recently modified by Dr. WILLIAMS. The mixture, as set down, is cloudy, and his modification consists in adding a certain amount of glycerine or syrup, namely, 1 to 2 drachms. This enables the mixture to keep clear for weeks. It should be diluted two or three times, otherwise the patient complains of burning pains. I give the mixture (℥i) three times a day, at eight, noon, and night. The important drugs in the mixture are iodide of potassium and tartar emetic. These two have a specific action—I do not think there is any doubt about it—on the spirochaete of yaws. As regards salicylate of soda, it has no action whatever on the spirochaetes and on true yaws lesions, but apparently it has a beneficial effect on the secondary pyogenic lesions which are so often seen in yaws; the thick crusts disappear much more quickly than when it is not given. Of this mixture one gives one ounce three times a day, and in this way one gets into the patient an enormous amount of iodide of potassium and tartar emetic, from which I have never noticed any bad effects, though occasionally slight signs of iodism may be observed. Apparently the presence of large doses of bicarbonate of soda prevents severe symptoms of iodism, and prevents also the emetic action of the tartar emetic, at least to a great extent. For adults I give full doses, but for women and children half to one third of a dose is enough. To Europeans it is better to give half doses; they do not stand full doses so well as do natives.

The results I have had have been very satisfactory, as you will have judged from the photographs I have shewn you. The treatment is peculiarly satisfactory in recent cases; indeed, it can be compared with the results following the intravenous injection of salvarsan. In subacute and chronic cases the results from my mixture are not so good. Still even in those cases the results are much better than after any other treatment I know, except salvarsan.

In conclusion, I would like to repeat that the salvarsan treatment is

FIG. 1.—A Case of Yaws

FIG. 2.—Same case after ten days' treatment with the mixture

To accompany Dr Castellani's article

the best we have for yaws; but when for any reason that treatment cannot be carried out, the mixture of which I have given you particulars will be found very useful.

Sir RONALD ROSS: Have you tried it for anything except yaws?

Major ALDO CASTELLANI: Yes, for tertiary syphilis, kala azar, bronchial spirochætosis and for relapsing fever, but the results are not so striking as those you see in frambesia. In tertiary syphilis, however, the beneficial effect of the mixture is apparent in a very short time. I have not tried the mixture for malaria, but I have tried tartar emetic for it, given intravenously. I have also given it by the mouth, mixed with quinine. I do not think tartar emetic has a specific action on the parasites of malaria, but I think that, like arsenic, phosphorus and other drugs, it may indirectly have a beneficial effect. I do not know how to explain it, but one certainly comes across cases in which the administration of arsenic or tartar emetic or phosphorus seems to increase the action of the quinine, though these drugs given alone have no effect.

TWO HELMINTHOLOGICAL NOTES.

By R. T. LEIPER, M.B., D.Sc.,

Helminthologist to the London School of Tropical Medicine.

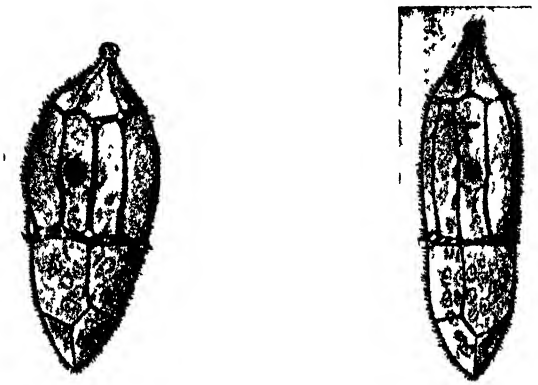
A. Note on the Integument of the Bilharzia *miracidium.*

Some months ago Dr. J. H. ASHWORTH, F.R.S., kindly sent me a slide shewing a number of miracidia of *B. hæmatobium*, prepared by him several years ago from a case of hæmaturia under treatment in Edinburgh. The specimens had been fixed in corrosive sublimate and then stained with hæmatoxylin. They now shew practically no internal details, but give a beautiful picture of the skeletal thickenings of cuticle supporting the integument. Two specimens have been drawn and illustrate this note.

Much has been written on the anatomy and developmental significance of the internal structure of the miracidia of bilharzia, but the cuticular arrangement so clearly shewn in these preparations has not so far been recorded. BROCK, LOOSS and LORTET noted two transverse zones of cuticular points jutting from amongst the cilia. From the accompanying figures it is seen now that these points are set in two rings of cuticle which divide the body of the miracidium into three zones. In the middle zone, six or seven longitudinal cuticular strands unite the two transverse rings, and divide the surface into rectangular areas. In the cephalic zone, cuticular strands radiate from the cuticle-covered rostellum to the anterior ring to form triangular areas. In the caudal zone, the areas formed by the cuticular strands are pentagonal, but are varied and irregular.

These areas correspond, I suppose, to the outline of the epithelial cells. In *Fasciola hepatica* these cells are described by LEUCKART as

polygonal. LORTET states that in bilharzia they are of similar shape, but that the silver method failed to bring their outlines into prominence.



B. A Simple Method for the Preservation of Helminth Ova in Fæces.

For class purposes, fæces containing the eggs of helminths are commonly preserved by the addition of five per cent. formalin. This is, however, an unsatisfactory medium, especially if it is desired to make permanent microscopical preparations. A modification of Looss' alcohol method has recently been published by STEPHENS in the new English edition of BRAUN'S *Animal Parasites of Man*. The fæces are poured into a solution of 70 per cent. alcohol. After sedimentation the alcohol is replaced hourly by fresh solutions of 70 per cent. alcohol to which 5 per cent., 10 per cent., and then 20 per cent. glycerine have been added. The final mixture is then exposed until the alcohol-and-water has evaporated, and a few drops of glycerine are added from time to time until the ova are in pure glycerine. Permanent microscopical preparations are made by transferring some of the sediment to glycerine jelly.

This method gives good results but is unnecessarily slow and complicated. I have found it practicable to shorten this considerably. Alcohol, 70 per cent., with 5 per cent. glycerine added, is raised to boiling point. Fluid fæces—or fæces made fluid by dilution with normal saline—are poured into the boiling alcohol, stirred, and then set aside to inspissate in a warm place. When the fæces have become a sticky mass, sufficient pure glycerine is added to make a soft paste.

Another procedure, which I have tested for the last three years, is simpler and as effective. It provides permanent mounts in glycerine jelly, and the eggs retain their original transparency and contour. To any quantity—say, half an ounce—of fluid fæces an equal bulk of Langeron's Lactophenol is added. The whole is intimately mixed by vigorous shaking. This mixture will form a pasty jelly, and will keep indefinitely. A permanent microscopical preparation can be made at any time by stirring a small portion into a drop of melted glycerine jelly on a slide, a cover-glass is placed on the jelly, and when this is set the preparation is completed by sealing with gold size.

Langeron's Lactophenol consists of carbolic acid one part, lactic acid one part, glycerine two parts, and water one part.

The fæces used should be of a creamy consistence. If solid they should be diluted with normal saline.

A CASE OF SPRUE FROM MECCA.

BY

NOEL E. WATERFIELD, M.B., B.S. (LOND.), F.R.C.S. (ENG.).

The patient, a native of Hadramut, who had been living in Mecca for some years, came to me at Port Sudan for treatment in March of this year. He complained of loss of flesh, frequent stools and soreness of the tongue. These symptoms had been noticed for three months or more. The patient was a thin, pale, wasted man of sixty; his mouth was full of septic stumps, and there was much pyorrhœa alveolaris; the mucous membrane of the tongue was glazed and bare of papillæ; the stools—of which he passed 10 to 12 in the twenty-four hours—were greyish, frothy and copious. No amœbæ were seen in the stools on microscopic examination.

A report from the Wellcome Research Laboratories—for which I am indebted to Dr. CHALMERS, the Director—stated that a pure growth of yeast, a variety of *Monilia enterica*, was grown from the saliva and fæces.

Treatment consisted in the removal of all septic stumps and carious teeth, administration of intestinal antiseptics, salol, and beta napthol in a bismuth mixture, and strict confinement to a milk diet with an occasional orange, the amount of milk taken being five pints in the twenty-four hours.

Within four days the diarrhœa stopped, and the patient's aspect changed for the better.

He left the hospital in a fortnight, as he regarded himself cured. In this time he put on nearly half a stone. He occasionally suffered from constipation, for which he took oil made from native butter.

I heard six weeks later that the cure appeared permanent, and that he was so pleased with the state of his health on the milk diet that he refused to try anything else.

The interest of the case lies in the facts, (1) that what was probably the causal agent was found in pure culture in the saliva and stools, and (2) the rapid cure after (*a*) the removal of the septic stumps and (*b*) the confinement to a milk diet.

NOTES ON A CASE OF ULCERATING GRANULOMA TREATED BY INTRAVENOUS INJECTIONS OF TARTAR EMETIC.

BY

E. N. DARWENT, M.D. (EDIN.),

Government Medical Officer, Arima, Trinidad.

On the 9th March, 1917, a young negro man was admitted to the Arima District Hospital, Trinidad. He was suffering from elephantiasis of the penis and scrotum and granulomatous ulceration in the pubic region, extending down either crutch and affecting the skin of the scrotum on either side, also the front of the scrotum and under surface of the penis where these meet. There was also a ring of ulceration around the prepuce.

I circumcised him on the 11th March, and put him on antisyphilitic treatment on the 16th March. As there was no sign of improvement I injected tartar emetic, one-third of a grain in 10 c.c. of normal saline solution intravenously, and applied caustic to all the ulcerated parts. I repeated the injection of one-third of a grain of tartar emetic intravenously on the 18th, 20th, 22nd, 24th and 26th March. There was now marked improvement. All the ulceration was healing, the raised edges had disappeared, and there was no pain. On the 29th March I injected half a grain of tartar emetic intravenously, and repeated this on the 31st March and 2nd April. By this time all the ulceration had healed soundly. He had a slight attack of diarrhœa, which lasted only a couple of days. On the 10th April I injected one grain of tartar emetic intravenously, and the patient was discharged well on the 13th of April, 1917.

CAMPING IN THE TROPICS.

BY

SIR DAVID BRUCE. C.B., F.R.S., F.R.C.P.

Surgeon-General, Army Medical Service.

1. THE TENT AND ITS ACCESSORIES.

At the present time, when many of our soldiers are doing duty with expeditionary forces in the tropics, the following simple hints may be of some service.

For the avoidance of mosquitoes and flies generally it is a comfortable and useful plan to make the verandah of the ordinary double-roof ridge tent into a fly-proof room. The following drawings illustrate this and render a detailed description unnecessary

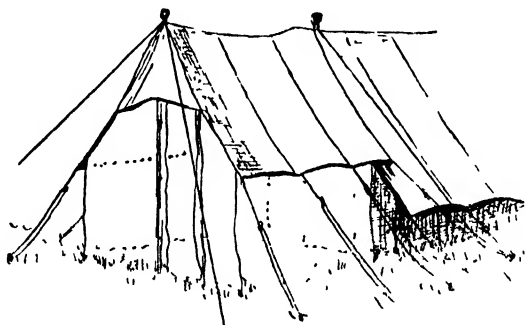


FIG. 1.—Fly-proof Verandah, down.

FIG. 2.—Fly-proof Verandah, up.

The net is attached permanently to the tent and can be fastened up out of the way during the day by means of tapes. At the lower edge of the net there is a wide edging of thin calico which can be weighted down by gun cases and chop boxes, so as to effectively seal up any stray openings. At the four corners are loops for tent-pegs. The doorway has three over-lapping layers of net, so that things can be passed through without much danger of insects finding their way in at the same time. The side of the fly-proof room opening into the tent itself is the same as that described, that is to say, it has a triple doorway, in order to prevent flies finding their way into the verandah from the tent. At meals one servant remains inside to receive the dishes, which are passed carefully through the triple doorway.

This fly-proof room is a convenient and practical arrangement, and enables the traveller or sportsman to read or write after sundown absolutely protected from mosquitoes or other insects.

The photograph shews a camp near Lake Nyasa. The mosquito curtains attached to the verandah are tied up, and it can be seen how little they are in the way during the day.

In addition to the fly-proof verandah it is necessary to have mosquito nets over the camp-beds. As a rule, these are held up by wooden stays, or a round hoop suspended from the roof, and are tucked under the

mattress. This is an untidy method, and it is much neater and takes up less room to have a net made to fit the shape of the tent (Fig. 4).

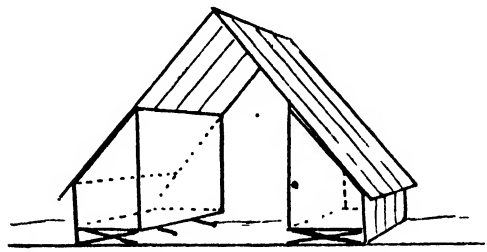
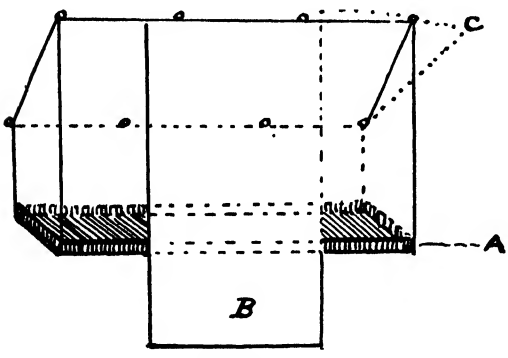


FIG. 4.

Strong hooks are sewn on the seams of the tent and eyes on the mosquito net. It is the simplest thing in the world for the native servant to slip the eyes over the hooks.

Another improvement is to make the net into a closed bag by sewing it to a floor or base of calico (Fig. 5).



A.—Calico floor to mosquito net.

B. Overlapping curtains of net, with calico ends for tucking in.

C.—Eyes for hanging net on hooks in tent.

FIG. 5.

The calico passes up the sides of the net for about six inches in order to strengthen it. The net in front is double for about three feet, the two curtains overlapping. This forms the opening into the mosquito-net, through which it is possible to slip without at the same time allowing mosquitoes to slip in too. The overlapping parts of the net have a wide margin of calico which can be tucked under the mattress after getting into bed.

The mattress is, of course, placed inside the net, resting on the calico base or floor, and the bed made up. When once inside and the calico

ends of the curtains forming the door tucked in, the traveller is safe from mosquitoes, ticks or ants. The interior is also roomy, the sides tight and trim, and altogether it is the easiest managed, the safest and the most comfortable net for a camp bedstead. It is well to have an electric lamp inside in case of alarm during the night, and, if thought necessary, a rifle may also be kept inside, lying along the edge of the mattress.

If there is any danger of mosquitoes being in the tent—and it is difficult to keep them out—it is well to get straight into bed from the mosquito-proof verandah and undress inside the mosquito net.

There is usually no difficulty in this, as dress in the tropics, as a rule, consists only of a khaki shirt and shorts. Talking of dress, I found a loose khaki shirt, only reaching as low as the hips and worn outside the shorts, to be cool and comfortable, If the shirt is made low and open at the neck there is a pleasant current of air set up from below upwards, which is comforting on a hot march. Some people try to arrive at the same end by wearing an ordinary shirt outside their trousers, but this is not nearly so neat and efficacious a plan. Of course, in tsetse-fly country shorts should not be worn, on account of the danger of being infected with sleeping sickness.

Another small improvement on the ordinary double-roof ridge tent may be mentioned. In the front wall or doorway there is an opening or window about a foot square. This is closed at night by a roll of canvas, which is let down and fastened *on the outside*. Of course, the reason for fastening the flap on the outside is to keep out rain and wind, and, if there is no verandah, doubtless this is an advantage. But if fastened outside, and anything startling occurs during the night, such as a lion grunting close by, it is impossible to look out without first unfastening the door and going outside to unfasten the window curtain. If the flap of canvas is removed from the outside to the inside it will be found more convenient. This was pointed out to me by Major PEARCE, Nyasaland. He also advocated the making of holes for ventilation in the inner canvas just under the ridge-pole. No doubt, a tent when securely shut up at night to keep out wild animals is badly ventilated.*

Another useful article in camp life is the thermos flask. When

*If anyone thinks it worth while to have a tent fitted up as described, I may say that Edgington, Duke Street, London Bridge, made me such a one in 1911.

travelling in the tropics, it is a good thing to get away from the camping ground as soon as it is light enough to see. If the cook is expected to make tea or coffee and supply an early breakfast it spells delay. The best way is to have the thermos flask filled at night with hot coffee, milk and sugar. The next morning a couple of raw eggs are beaten up in a cup and the coffee added. This is swallowed in a few seconds, and makes an excellent early breakfast. The cook does not require to make a fire, and he can have his pots and pans packed up the previous evening.

As a rule the cook and his assistants are left to pack and follow. This is a mistake. They dawdle along the road, talking to every native they meet, and are never at hand when wanted. The whole column should be started at once, and rigid discipline preserved until the evening camp is reached. The leader walks at the head of the column and his lieutenant at the rear. If any man leaves the column the lieutenant blows a whistle, the whole line is brought to a halt and the man brought back. If he has no reasonable excuse, he is reprimanded, ordered back into his place, the whistle is blown and the column again takes up its march. In a few days the men learn the rules and no one leaves his place. If this is not done the men straggle along the path, visit villages, get beer, and turn up in camp hours late. The man who has strayed is usually carrying some important part of the tent or equipment, and nothing can be more annoying than to wait an hour or more for some such laggard to turn up with the tent poles or pegs. But by keeping up rigid discipline the men all march into camp at the same time, the tents are pitched in a few minutes, and the men are at liberty to feed, rest and amuse themselves as they think fit. Of course, suitable halts are made. After the first mile or two, when it is fully light, a halt of ten to fifteen minutes is made, to enable the porters to readjust their loads, and at least five minutes every hour is given as a breathing space. About ten or eleven o'clock a long halt is made for breakfast. Table and chairs are set up in a shady place, a fire lit, and a good breakfast cooked and eaten at leisure. Before starting again the cook fills the thermos flask with hot tea, milk and sugar, places it in the luncheon basket, and so afternoon tea can be had immediately on reaching camp. After such a march most people are thoroughly tired and wearied, and the immediate tea is very grateful. The cook and the other servants can also be set to help to pitch the tents instead of starting to light fires and unpack the kettle.

2. PROTECTION FROM TSETSE AND OTHER BITING FLIES.

When shooting or working in a tsetse-fly country some protection from the bites of these poisonous insects is necessary. It must be borne in mind that a single bite from an infected tsetse-fly will almost certainly give rise to sleeping sickness. It is evident, then, that shorts are not permissible. Their place must be taken by strong drill khaki breeches, with boots and gaiters or puttees. It is best to wear a coat over the shirt in order to protect the body and arms. The neck and face are favourite places of attack, and anything in the way of a net veil or covering attached to the brim of the helmet is apt to be hot and stuffy, and, further, is constantly being caught and torn by the thorns with which all the trees in the fly-country are plentifully supplied. Moreover, the veil interferes with vision, and is in the way when shooting.

FIG. 6.

To remedy this a headpiece was invented, which was found to be practical, fairly cool and safe (Fig. 6). Knitting-needles, about one-third of an inch in thickness, were made of hard wood. With these a head-covering was knitted, similar in appearance to a Crusader's headpiece of chain-mail. Native string, which is stiff and hard like whipcord, was used. The result was a close-fitting cap, extending well down on the

chest and back, and leaving only a small opening for the eyes, nose and mouth. It was found that the tsetse-flies seldom attacked this opening, and, if they did, were seen and driven away. The string made a layer about one-third of an inch thick like chain-mail, full of spaces and interstices, which permitted free ventilation and evaporation. But a tsetse or other biting fly could get through these spaces. To prevent this a layer of coarse mosquito netting was sewn on the surface of the knitted string, so that a fly found its proboscis too short to reach the skin. As this fly-proof helmet lies close to the head, it is possible to creep or push through thorny scrub without any danger of being held up. Of course, as a protection against the sun, a large felt or terai hat must be worn over the helmet, but this can easily be arranged.

When cycling through fly-country, especially on a motor-cycle, the tsetse-flies often attack in great numbers, and very viciously. Sometimes they follow like a swarm of angry bees, and it is necessary to stop now and then and break off a branch in order by its aid to drive them away. On these occasions the cyclist is liable to be severely bitten unless he is adequately protected. A net is of little use, but the above-described headgear, with the addition of a pair of motor-goggles, gives excellent protection for the face and neck.

It is necessary to see that there is no undefended space between the goggles and the helmet. The only parts exposed are the nose and mouth; if a fly attacks here it can be blown away.

To protect the hands, fingerless gloves with khaki gauntlets were made in the same way, except that the palm was made of leather, with a hole for the forefinger to go through.

3. PROTECTION FROM MOSQUITOES FOR SOLDIERS ON ACTIVE SERVICE IN MALARIOUS COUNTRIES.

This is an important and difficult subject. Important, because many campaigns have been rendered null and void by the barrier of malaria. Armies armed to the teeth with quick-firing guns, and all the panoply of modern war, may just as easily be routed by the minute anopheles mosquito as were the older armies. Difficult, because it is difficult so see how soldiers on active service can be protected from mosquitoes. They have often to march long distances in great heat and with a heavy kit to

carry. Naturally they want to expose as much bare skin as possible for evaporation purposes. Hence the use of shorts and shirt with rolled-up sleeves. The malaria-carrying mosquitoes bite at night, it is true, but this does not help much, as marching must often be done at night, and, even if men are in camp, it is almost impossible to escape being bitten during the evening or night. Much can doubtless be done by a wise selection of camping grounds, but the question to be answered is, Is it possible to provide a soldier on active service with a practicable protection against mosquitoes? As this is a subject of which I have no practical experience, I must leave the question to others to answer.

Professor SIMPSON has lately demonstrated to the Society several ingenious methods of protecting against mosquitoes; it is hoped that these few remarks on camping in the tropics may form a small addition to the subject.

Obituary.

JOHN MITFORD ATKINSON, M.B. (Lond.).

We have with great regret to record the death of one of our Fellows and Member of Council, John Mitford Atkinson, M.B. (Lond.), who took a great interest in and contributed much to the extension and success of our Society in China.

This was natural in one so deeply interested in tropical medicine, and who did so much towards its progress, both in its medical and hygienic aspects during a long residence in Hong Kong.

In 1887, Dr. Atkinson was chosen out of a number of candidates for the appointment of Superintendent of the Government Civil Hospital, Hong Kong, having previously been for seven years resident physician to the Kensington Infirmary. On his arrival in the colony he at once devoted himself with characteristic energy to making his hospital equal to the best managed of our home hospitals.

The corresponding status as between the Colonial Surgeon and the Superintendent Civil Hospital had been rather indefinite, and occasionally led to friction, but Dr. Atkinson managed by his keenness and evident ability to get the control practically into his own hands, and on the retirement of the Colonial Surgeon in 1897 was appointed Principal Civil Medical Officer, and thereby chief of both the Medical and Health Departments, and given a seat on the Executive Council of the colony.

One of his first acts on arrival was to induce the Government to bring out a staff of trained nurses from England, and he naturally sought them in his old school, "the London." In 1890 Miss Eastmond (later to become Mrs. Atkinson) came out as matron, with six sisters. With this able assistance Dr. Atkinson soon produced a marked improvement in the condition, discipline and general management of the hospital, a noticeable feature being the attention paid to the private wards, there being at that time no other suitable

quarters for the stranded sick Europeans, and more especially the wives of the lower grade of Civil Servants.

The accommodation for lunatics, both "foreign" and Chinese, left much to be desired, and here, too, Dr. ATKINSON'S energy and attention, in conjunction with the Colonial Surgeon, Dr. AYRES, soon wrought a great change for the better. In the Chinese Hospital (the Tung Wa) lunatics were confined in dark cells without windows, the only light filtering through a barred gate, never cleaned, and bad food being given at very uncertain intervals. After strong representations to the proper Chinese authorities, who were nominally under the superintendence of the Colonial Surgeon, but whose advice "not being backed up" they had hitherto ignored, their condition was gradually improved, and the Tung Wa itself was later "brought into touch" with the Civil Hospital.

In 1894 and 1896 Hong Kong was visited by very severe epidemics of bubonic plague, for which many special mat-shed hospitals were opened. One of the largest was in connection with the Civil Hospital, and although all of us medicos—naval, military and civil—lent a hand, a very heavy responsibility and arduous extra duties were thrown on Dr. ATKINSON. For his services he received the thanks of the Government.

There have been many recurrences of plague in Hong Kong since but nothing equal to these, when the mortality was nearly 90 per cent., and the hospitals so crowded that you had to pick your way through the patients lying on the floor, and numbers of Chinese dead and dying were being dumped in the streets. Two of the Civil Hospital sisters died, and the rest of the staff, including their Superintendent, were "run off their legs."

The men of the Russian cruiser "Variag," wounded in the battle with the Japanese near Chemulpo, were admitted to the Government Civil Hospital, and for his services to them Dr. ATKINSON received from H.M. the Czar a gold cigarette case with the Russian coat of arms in diamonds.

Dr. ATKINSON was mainly instrumental in having the Victoria Jubilee Hospital opened at The Peak, principally for maternity cases, both Government servants and private. It has proved a great boon to the former, enabling them to escape from the great heat of the town.

Dr. ATKINSON'S annual reports, first as Superintendent Government Civil Hospital and later as Principal Civil Medical Officer, were most valuable, and models of what such papers should be; not mere pages of dry statistics but well-thought-out treatises, full of interesting information on local conditions and the tropical diseases dependent thereon. He was an active member of the Hong Kong and China Medical Society—later to become a branch of the British Medical Association—of which he was president, and afterwards represented the branch on the General Council of the British Medical Association. He was also a keen cricketer, and encouraged a healthy interest in outdoor games amongst the younger members of the Civil Service.

In 1912 Dr. ATKINSON, whose health latterly had not been very good, took his well-earned pension, but did not "go out of harness." He "set up his plate" in London and was gradually building up a successful tropical practice. He was elected a member of the Council of this Society in June, 1915.

At the outbreak of war he offered his services, and was given charge of one of the large military hospitals at Richmond with the rank of Temporary Major R.A.M.C. The work was responsible and trying, with long and uncertain hours. In addition to our own wounded, a large number of German prisoners were under his care.

He was also a representative of the Hong Kong War Charities on the Committee of the Royal Flying Corps Hospital, where his advice and assistance were much valued, and suitably acknowledged by Lieut.-Gen. Sir DAVID HENDERSON.

Even failing health did not lessen his interest in work, and Fellows will remember the valuable contribution he made to our TRANSACTIONS on cerebro-spinal fever. Those of us who were present little thought that the alert, soldier-like figure was so soon "to pass beyond the border." Renewed attacks of angina compelled him to resign his commission. Though a short holiday with rest improved his health, the attacks becoming less frequent, they did not cease, and the end came suddenly and peacefully on May 23rd.

To his sorrowing widow and two sons, students at St. Paul's, we offer, on behalf of the Society, our sincerest sympathy. W. H.

JOHN CROPPER, M.A., M.D. (CANTAB.), LIEUT. R.A.M.C.

We regret to record the death of Dr. JOHN CROPPER, a Fellow of the Society, who was lost at sea when the hospital ship "Britannic" was sunk in the Ægean, in November, 1916. Dr. CROPPER, who was fifty-three at the time of his death, was a graduate of Trinity College, Cambridge, and studied at St. Bartholomew's Hospital. In 1895 he went out to Palestine to the Church Missionary Society's station at Acre, and, in order to fit himself for his work, he took the Turkish medical diploma by examination at Constantinople. In 1900 he resigned for family reasons, but in 1903-6 he was again in Palestine, now at Ramallah, near Jerusalem, in temporary charge of medical mission work. All his work for the C.M.S. was honorary. Dr. CROPPER contributed two interesting papers to the *Journal of Hygiene* on Anopheles and Malarial Fever in Upper Palestine and Jerusalem (1902 and 1905). He reported on many of the Jewish colonies, among which malaria and blackwater fever were prevalent. He shewed that though Jerusalem is practically without natural water supply, malaria is rife, the anopheles breeding in the cisterns. Dr CROPPER has contributed to the discussions at the Society's meetings, and in 1908 demonstrated blood-slides shewing phenomenal abundance of parasites in the peripheral circulation of a fatal case of pernicious malaria in Palestine. He married in 1895, and when the war broke out was practising at Chepstow. He then took up Red Cross work in France, and later joined the R.A.M.C. From a notice in the *Church Missionary Review* we learn, "It now seems certain that he sacrificed his life by giving up his life-belt to a ship's stoker who was without one."

ELECTION OF FELLOWS.

The following were duly elected Fellows of the Society :—

T. W. Hoggarth, M.D., Melbourne.

Captain J. T. Edwards, B.Sc., M.R.C.V.S.

EXCHANGES.

Anales de la Academia de Ciencias Medicas Fisicas y Naturales de la Habana.
Annali di Medicina Navale e Coloniale.
Annals of Tropical Medicine and Parasitology.
Archives de l'Institut Pasteur Tunis.
Arquivos do Instituto Bacteriologico Camara Pestana.
Bulletin Public Health and Marine Hospital Service of the U.S.
Bulletin of the Tropical Diseases Bureau.
Bulletin de la Société de Pathologie Exotique.
Bulletin de la Société Portuguaise des Sciences Naturelles.
Bulletin de l'Academie Royale de Médecine de Belgique.
het Geneeskundig tydschrift voor Nederlandsch-Indie.
Indian Journal of Medical Research.
Indian Medical Gazette.
Johns Hopkins Hospital Bulletin.
Journal of the Royal Naval Medical Service.
Journal of the Royal Army Medical Corps.
Journal of Tropical Medicine and Hygiene.
Journal of Tropical Veterinary Science.
L'Enseignement Medico Mutuel Internationale.
Memorias do Instituto Oswaldo Cruz.
New Orleans Medical and Surgical Journal.
Philippine Journal of Science.
Revue de Médecine et d'Hygiene Tropicale
Sanidad y Beneficencia.
Veterinary News.

NOTE.—The above publications lie in the Society's room at 11, Chandos Street, W., 1, and are at the disposal of Fellows.

J. C. PHELP AND SON, PRINTERS, BEULAH ROAD, WALTHAMSTOW.